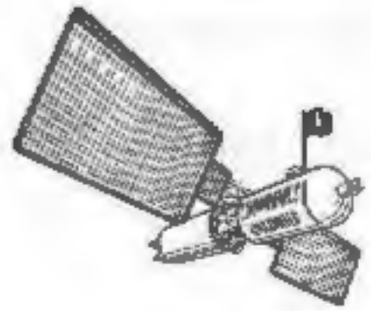
SILENT DEATH
Revised & Expanded
2nd Edition

by Uncle Fester



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Contents

Dedication

This book is dedicated to my enemies, without whose cooperation it would not have been possible.

PREFACE

In earlier, simpler times, when men lived in small bands close to nature, there existed in each band a very respected and special individual to whom was entrusted the ancient knowledge of the healing and other special properties of the plants which grew in the area. This person was the shaman or medicine man of the tribe, and it is one part of his craft which we will celebrate in this book. This book is a celebration of that ancient and fine art, the art of poisoning.

The advance of medical and chemical technology in recent times has made successful poisoning more difficult than it was in the days of the shaman. The simple and easily available poisons such as rat poison or arsenic can all be easily detected in the body of the victim if a thorough autopsy is done. In the case of inorganic poisons such as arsenic, mercury, cadmium, etc., the tell-tale traces of these substances can be found in the body of the deceased for as long as the remains are around to be analyzed. It makes no difference if the body is embalmed or cremated, for these substances are chemical elements and can't be destroyed by anything short of nuclear hellfire. For this reason, the successful poisoner must avoid such quick fixes and return to his cultural roots, the knowledge of the shaman, if he is to avoid detection.

It is a sad commentary on the brutish times we live in that the use of deadly substances as a means of homicide is virtually unheard of. Instead of the quiet dignity of an effective poison, those with

homicidal intent seem to impulsively reach for a gun, knife or club. All these crude instruments leave no doubt as to the cause of death. As an old associate of mine once said, "What are we, swamp animals or technological warriors?" I am afraid that, in all too many cases, the answer is the former.

This book will attempt to cover, in simple language, the many aspects of successful poisoning. From the crude inorganic (i.e., mineral based) poisons which get so many people a one-way ticket to the Big House, to the much more subtle and difficult to detect organic poisons, we will explore the methods used by artists skilled in the craft to avoid detection. We will also cover the procedures used in forensic toxicology labs to detect the presence of toxic substances in the body of the victim. For those really big jobs, we will cover in detail the production and use of some of the more deadly war gases. These gases were so horrible when they were used in World War I that they have not been used in a major war since then. For those who have whole armies to conquer single-handedly, I'm sure the section on the production and use of nerve gases will interest you. These nerve gases have been called "the poor man's atom bomb," with good reason. They make the World War I war gases look like kids' stuff. I'm sure you'll be surprised how easy to make and use these little gems are. Read on and enjoy...

Chapter One
WHAT MAKES A GOOD POISON?

When considering the large number of substances which can exert a deadly effect on the human body, the question naturally comes up, "What makes one substance a good poison, and another one not so good or actually bad?" The answer to that question, of course, depends upon the circumstances in which the poison is to be used. What may be an excellent war gas may not be very suitable to the assassination of one person, and vice versa. Choosing the right poison for the job is like calling the right play in football. It requires knowledge of the subject and use of psychology to make the right choice. There are, however, some general rules of thumb to follow which make the subject considerably simpler.

The first two general rules are the golden rules of rat poisoning, which is only right, since most targets are rats anyway. Rule number one is that the little goodie to be delivered to the subject must not be overly unpalatable. This rule, of course, is just common sense, but it is also the most violated rule of good poisoning. It is unbelievable how often people do stupid things like put lye or cleaning fluid in an intended target's coffee, just to have it spat right out. You can imagine the kind of scenes this blunder can lead to, especially when the would-be poisoner is serving the drink. A great deal of embarrassment can be avoided by paying some attention to how the intended target is likely to react to the deadly substance being offered. If the poison is going to be administered by mouth, it may be wise to choose one of the

tasteless, odorless poisons described in this book. Alternatively, some of the brew may be mixed up beforehand, and a small drop of the mixture tasted. This may at first glance seem foolhardy, but so long as it is immediately spat out and the mouth thoroughly rinsed, it poses little danger. A good mix for really foul-tasting poisons is a shot of whiskey. More on this later.

The second golden rule of rat elimination says that the material should not cause bait shyness before a lethal dose is consumed. Several factors come into play here. Most important is the length of time between consuming the little goodie and the onset of the symptoms of poisoning. For example, let's say the mark is a slow eater, and is being given one of the materials which shows some early effect such as numbness or burning of the mouth soon after any is consumed. This could lead to failure of the mission, and the most dire consequences for the would-be poisoner. This problem becomes very tricky when a large group is to be attacked at once, since people naturally become very wary when they start to see their associates keeling over.

I see several ways around this problem. First of all, one of the slow-acting poisons such as the jequirity bean or botulin may be served. As an alternative, one of the many fine gases described in this book may be used. I would especially recommend phosgene, arsine and phosphine in this respect, because they don't have much odor at lethal concentrations, and their effects are delayed so that an entire group can be taken out if so desired.

The third general rule of good poisoning is that how quickly a poison acts is crucial to its value in any mission. To my way of thinking on this subject, a poison should either cause death very quickly before help can be reached, or its action should be very much delayed to cause the victim to be unable to pinpoint the cause of his distressing condition. In this respect, I feel some of the cancer-causing agents covered in this book are very appealing. Their use requires patience, but the knowledge that a time bomb has been planted in the mark and is ticking away can be very satisfying.

The fourth general rule of good poisoning is that the material should be available without leaving a trail of suspicious purchases to lead the finger of guilt pointing back to the perpetrator. For this reason, poisons which can be obtained from plants are emphasized in this book. Many very beautiful plants have very deadly effects. Growing a few of them would raise absolutely no suspicion. In addition, there are many wild plants which are also deadly poisonous. A good field guide book, available at any library or book store, would reveal to the reader a whole new world of which he was previously unaware.

It is also important that the material be locally available. I can see little value in a substance which one must travel half way around the world to obtain. In this same vein, there are many fine deadly substances which can easily be stolen or "borrowed" if a person knows where to look for them. See the list at the end of this book of the chemicals mentioned in this book along with their major industrial and commercial uses for hints on where to look for these deadly substances.

The fifth and final general rule of good poisoning dictates that the substance used should be very difficult to detect in the body, both before death, to frustrate treatment, and after death, to frustrate the ensuing investigation. To make this easier, the symptoms and appearance of the body should mimic one or more natural diseases, so that death may be attributed to this disease at autopsy (see Chapter Thirteen). It is also important that the substance being used is effective in very small amounts, so that its presence does not jump out at the person doing the lab tests at autopsy. Much more on this later, but suffice it to say now that if the substance is exotic, it is not likely to be looked for in the corpse at autopsy, and if they do not look for it, it will not be found. Along this line, I once again have to sing the praises of the time-delay poisons. These wonderful little gems, like the cancer-causing agents which can be extracted from plants, or obtained from industrial sources, or cooked up on your own, guarantee almost complete safety of the poisoner. So long as he does not boast of his cunning,

or get caught in the act, I see no way for his deeds to land him behind bars. After all, if the cause of an individual case of cancer could be proven, the tobacco companies would have been sued into bankruptcy years ago.

One quick word here before we move on. It is a natural human tendency to believe that if a little is good, more must be better. This could not be farther from the truth in the field of good poisoning. The human stomach is a sensitive organ and may very well rebel against being given a megadose of any substance. An untimely attack of vomiting could very well lead to failure of the mission, with the most dire consequences for those involved. A good general rule is to never use more than twice the recommended dose, unless the victim is very fat. There is an added benefit in this, as smaller dosages are more difficult to detect later.

Chapter Two
INORGANIC POISONS

Inorganic poisons, as was mentioned earlier, are mineral based substances capable of causing death. Most of these are what popularly come to mind when someone mentions poisons. Examples are arsenic, mercury, cadmium, and fluorides. These, as a rule, are really bad poisons, with a couple exceptions which will be examined in detail in this chapter. The best thing which can be said about these inorganic poisons is that they do the job well, meaning that the victim dies, and they are pretty easily available if you know where to look. On the bad side, they are too easy to detect, and the victim knows pretty quickly that he has been poisoned. It usually takes a good while for the victim to die, so he has plenty of time to say where he was served his fatal repast. Why even cover these inferior substances? Because a quick coverage of these bad poisons will help the reader to appreciate the qualities that a good poison has.

Fluorides

The fluorides are common, easily available chemicals with a lot of uses in industry and public health. Two of the most common fluorides are sodium fluoride, NaF, and sodium fluorosilicate, Na_2SiF_6. They look a lot like table salt, so their appearance does not cause alarm in the victim. After being served a fatal dose of fluoride, the victim begins to throw up and gets cramps. These

cramps worsen until the victim has convulsions and turns a blue grey color. Finally he dies, because fluoride stops the enzymes in the body from functioning. Think about that when drinking fluoridated water. The body gets rid of fluoride very slowly, so when the body is examined at autopsy, most of the original dose is still there to be found. A quick test for fluorine is a standard part of most every autopsy where poisoning is suspected. The fatal dose of sodium fluoride is about 5 grams, for sodium fluorosilicate the fatal dose is about 1 gram. They will have no problem detecting these quantities. You can read more about fluoride poisoning in *The American Journal of Medical Science*, Volume 197, page 625 (1939). The author is Gettler.

Heavy Metals

These materials have nothing to do with rock music. Instead it is a term used to refer to mercury, lead, cadmium and the compounds that they form. Since none of the heavy metals are normally found in the body in any large amount, finding them at autopsy will be evidence enough to prove poisoning. A test for the presence of heavy metals is a standard part of every autopsy where poisoning is suspected. Mercury is pretty representative of the whole group, so I will just describe its effects without getting into lead and cadmium.

Most of us are probably familiar with mercury metal, the silvery liquid that fills some thermometers. It is called quicksilver, and is not particularly dangerous, except for its vapors. That is because it does not dissolve into the water in the body, so it can't get around to do its work. The salts of mercury are much more poisonous, and of these, corrosive sublimate, $HgCl_2$, is probably the worst. A person who swallows some immediately notices a harsh metal taste in his mouth, followed by a burning feeling in the stomach. Soon after that, bloody vomit starts coming, and later, bloody diarrhea. The kidneys stop working, so the flow of urine comes to a halt. Death can occur within one hour from a big dose, but it is more

likely to occur days later from exhaustion. The fatal dose depends on how soon after eating the poison the victim begins to vomit. If vomiting does not occur, .2 gram will be enough to cause death. For more information, see *The American Journal of Medical Science*, Volume 185, page 149 (1933). The author is Peters.

Oxalates

Oxalates are compounds containing oxalic acid. These poisons are a lot better than the ones mentioned so far because oxalic acid is made from carbon and oxygen, two very common elements in the body. For this reason, they are not so bad as most of the rest of the inorganic poisons. A cremated body will carry no trace of poison in the ashes. In spite of this, it is still a bad poison because such a large dose is needed (about 10 grams) to cause death. It also leaves the kidneys plugged up with calcium deposits, and these will stick out like a sore thumb at autopsy. Oxalates are what make rhubarb leaves poisonous, so they are really easy to get hold of. Serving them is another matter. For more information, see *Journal of Physiology*, Volume 16, page 476, (1894), by Howell.

Arsenic

This old stand-by has long passed the time when it was of any use. It used to have going for it the fact that the victim could be slowly poisoned, making it seem that he was in the grips of some prolonged illness. Now, however, it is all too easy to detect arsenic in the body. The most deadly arsenic compound is arsenic trioxide, As_2O_3. It has a faint sweet taste, and a fatal dose of .2 gram.

Cyanides

These pack a lot of punch, and leave no doubt as to the outcome of the matter, but ever since the Tylenol murders, coroners have cyanide on the brain. A person who has been fed sodium or

potassium cyanide will have the characteristic smell of cyanide on their breath, not to mention more of it still in their stomach. The gas, hydrogen cyanide, is much less obvious or detectable, but it's not exactly subtle either. If you have read *The Poor Man's James Bond* and feel like fooling around with hydrogen cyanide (HCN), I must warn you that the apparatus shown for making hydrogen cyanide is not up to the task of liquefying the gas and keeping it a liquid until used. HCN boils at 26° C, which is normal room temperature. Even well below that temperature it evaporates off fumes like crazy. If you have ever fooled around with ether, you know what I mean. These fumes are very deadly, one part per 2000 air is a deadly concentration. If you must fool around with the stuff, *Organic Syntheses* reports that if you smoke a cigarette while around HCN, the taste of the smoke will become unpleasant if you are breathing in any HCN. The fatal dose of sodium or potassium cyanide is about .1 gram. For HCN it is about .05 gram (50 milligrams). This is a little over $^1/_{20}$ of a quart of gas, if inhaled directly.

Carbon Monoxide

After that brief coverage of some of the bad poisons, let's move on to the good ones. Carbon monoxide is a poison I can really get excited about, to paraphrase my favorite TV salesman. It's not that carbon monoxide is so hard to detect that makes it so good. On the contrary, it's pretty obvious. The beauty of carbon monoxide is that an attack with carbon monoxide can so easily be made to look like an accident. I live in the northern part of the country, and during the cold weather season it is a very common event for people to be overcome by carbon monoxide in their homes. This is usually because their furnace was not burning its fuel properly, or because a squirrel or some other creature decided to build a nest in the exhaust flue. These facts can be taken advantage of by the

resourceful poisoner to make an attack appear to be an unfortunate accident.

Carbon monoxide is a colorless, odorless, tasteless gas with the chemical formula CO. It is formed by burning materials when they do not get enough air to burn completely to carbon dioxide. It is also flammable, so care must be taken when handling large amounts of the gas.

Carbon monoxide is poisonous because it gets into the bloodstream where it combines with the hemoglobin in the blood. This combination is not able to carry oxygen anymore, so the victim suffocates. The first symptom of carbon monoxide poisoning is a headache, which is followed by weakness, nausea and dizziness, leading finally to a coma and death. The skin of the victim is cherry red, a sure sign of carbon monoxide poisoning.

There is a formula to use in calculating the fatal dose of carbon monoxide. It is pretty simple, but I will explain how to use it, for everyone who slept through math class. The formula is:

(hours exposure) (parts CO per 10,000)>15

This means that if the hours that the victim is exposed to carbon monoxide, times parts CO per 10,000 air is greater than 15, a fatal dose has been absorbed by the target. For example, let's say that the mark is in a trailer home (they have notoriously bad heaters) measuring 50 feet long, 15 feet wide and 8 feet high. This means that the trailer has a volume of 6000 cubic feet, because the formula for finding the volume of a box-like object is length times width times height. Plugging this figure into the original formula for a one hour exposure, we get 6000 divided by 10,000 times 15 equals 9. Any amount of carbon monoxide over 9 cubic feet is a lethal dose in one hour's time. With a two hour exposure, the lethal dose is anything over 4 and one half cubic feet.

It is very important here that the attacker resist the normal human impulse to put too much of the gas into the target. At autopsy, the amount of carbon monoxide in the blood will be

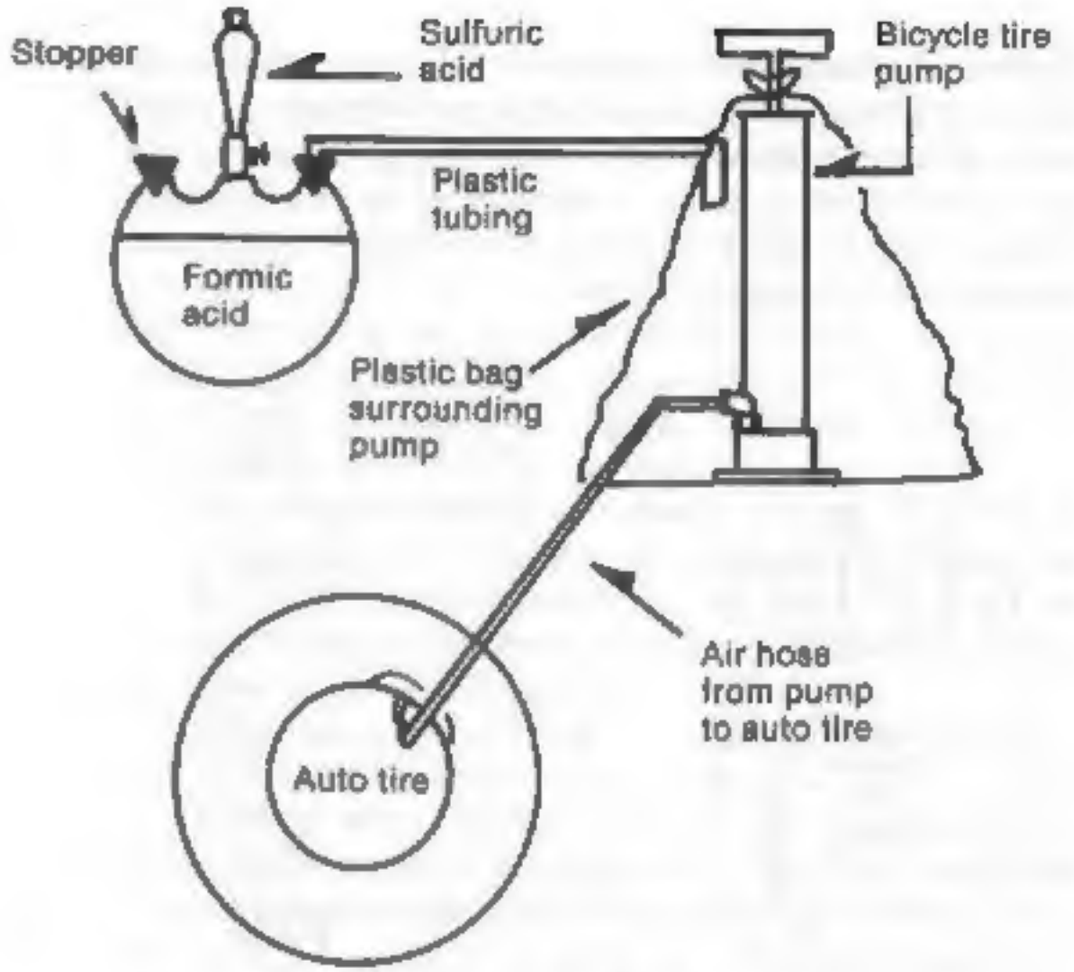
measured, and anything far above the lethal range will be cause for suspicion. I think that, taking into account circulation of fresh air into the target, not more than double the lethal dose of carbon monoxide should be used. If the target building is sealed up for cold weather, that amount should do the job well.

As you can see, carbon monoxide is a very sneaky and versatile poison. The question which naturally follows is: "How does the attacker deliver the carbon monoxide to the target?" The answer to that is that he has several options. The simplest, and in many ways the best course is for the attacker to simply plug up the exhaust flue for the target's heating system. This works best for natural gas heated buildings, less well with fuel oil heated targets, and of course, not at all for electrically heated buildings. If the target is electrically heated, the car attack scheme that follows may be resorted to. In plugging up the exhaust flue of the target, the attacker takes care to use things like dry leaves, plant stems, and small plastic objects. These are typical materials used by wild creatures to make nests for themselves in a warm place like an exhaust flue. This plan of action has the advantage that whatever tragic events ensue are all easily explained on investigation as simply an unfortunate accident.

If this subtle form of attack fails, the determined poisoner then must resort to a more direct attack. This is done by actually pumping some carbon monoxide into the target building. Here is how it is done:

The attacker rigs up the apparatus (shown on the next page) in a garage or similar building. He takes care to make sure that a fan is set up in a window, blowing outward, to keep carbon monoxide from building up in the building.

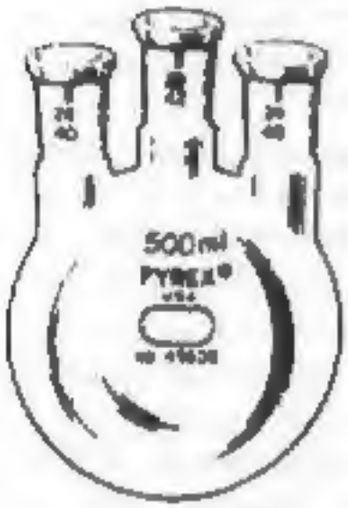
You see in the diagram on the next page, a system which can be used to make pure carbon monoxide, and then pump it into an automobile tire for storage and transportation. I will start by explaining the set of glassware seen in the upper left hand portion of the system, since this particular rig will be seen a few more times in this book.



Individual parts not drawn to scale

These two pieces of chemical glassware are very useful for making gases. They have ground glass joints which fit together tightly, preventing dangerous gas leaks. The valve on the separatory funnel allows chemicals to be run from the funnel into the flask without taking apart the rig, causing gas to spew out into the air. These two pieces of glassware run about $50 each, and are well worth it. To my way of thinking, no home is complete without some laboratory equipment laying around. These two pieces are easily available from scientific supply houses (my favorite is Sargent-Welch in Skokie, IL 60077). No suspicion will fall on a person who orders these two pieces of equipment, especially not

suspicion of poison gas manufacture. A very poor substitute for these two pieces would be a large champagne bottle with the plastic tubing connected to the top of the bottle. This is, however, a very poor substitute which makes a mockery of the deadly serious business which poisoning is. It also adds a large danger that the poisoner may become the poisonee.



3-necked flask

Separatory funnel

Now for how this system works. You see the separatory funnel in the diagram has been stuck into the middle neck of the 3-necked flask. For this connection to be air tight, the ground glass joints must be of the same size. My favorite size is 24/40, but any size will do. You will also notice that one of the necks of the 3-necked flask has been stoppered (a cork will do for this, some bubble gum will help seal it up), and finally, into the third neck of the flask, a

section of clear plastic tubing has been inserted. The diameter of this tubing should be big enough to fit tightly into the neck of the flask and form a tight seal. Some bubble gum can also help to seal this up.

Now notice the plastic bag that this tubing leads to. This bag surrounds a bicycle tire pump (or a foot type pump will work as well) which is connected to an automobile tire. It is very important that this bag not leak, and that the pump may be worked without wrecking the bag. This is all checked out *before* the determined poisoner starts to make carbon monoxide.

Finally, the automobile tire *must* have an inner tube in it. This is so that it does not leak while filling it, and also so that the attacker may start with a deflated inner tube. This makes calculating the amount of carbon monoxide in the tire much simpler than starting with a tire that already has air in it.

Now that all these things have been checked out, the poisoner is ready to start making carbon monoxide. Into the 1000 ml 3-necked flask is placed 500 ml of formic acid. The usual grade of formic acid is 88% pure, and works quite well. Into the separatory funnel is placed 100 ml of concentrated sulfuric acid. Good eye protection, like a pair of goggles, is always worn by the person using these chemicals, because strong chemicals like these can easily cause blindness if splashed accidentally in the eyes. With his eye protection in place, the gasser opens up the valve on the separatory funnel and lets about half of the 100 ml of concentrated sulfuric acid flow into the formic acid, and swirls it around to mix it into the formic acid. A little bubbling may start now, but as a rule, the mixture must be heated to get production of carbon monoxide going.

To do this, the 3-necked flask is put in a pan that is just a little bigger around than the flask is, the pan is then filled a little over half full of water, and the pan and flask are put on a single burner hot plate buffet range with infinite temperature control knob. One of these costs about $20 at the local hardware store. The heat is turned on to the pan, and the water is brought to a simmer. As the

acid mixture heats up in the flask, bubbling out of carbon monoxide gets going well. The reaction going on here is

$$HCOOH \xrightarrow{\text{sulfuric acid}} CO + H_2O$$

formic acid → carbon monoxide + water

Formic acid, when it gets heated, breaks down into water and carbon monoxide. Sulfuric acid acts as a catalyst for this reaction by soaking up the water formed in the reaction.

So as the carbon monoxide begins filling up the plastic bag, it is pumped into the automobile tire. This could require some fast pumping at first to keep up, but soon the sulfuric acid will have soaked up about as much water as it can, and the bubbling out of carbon monoxide gas will slow down. Then it is time for the poisoner to add the rest of the sulfuric acid from the separatory funnel into the flask, and close the valve.

The pressure in the tire must be followed closely after it begins to get to full pressure. From 500 ml of formic acid, up to 10 cubic feet of carbon monoxide can be formed. Now the volume of the average auto tire (15 inch rim, 8 inch diameter inner tube) I calculate at about 2.1 cubic feet. If it is inflated to 45 lbs. pressure, which I think is about the upper limit of safety, that means it is holding triple normal atmospheric pressure, and so means that it is holding triple the 2.1 cubic feet of carbon monoxide, or 6.3 cubic feet of carbon monoxide. This means that the tire will be pumped up to maximum pressure about halfway through the reaction, and a fresh one must be ready to go to hold the remaining carbon monoxide. The exact amount of carbon monoxide in each tire can be calculated by finding the volume of the inner tube, measuring the pressure of the gas in the tire with a tire gauge, and plugging these numbers into this formula

$$\frac{\text{pressure in tire}}{\text{15 lbs per sq. inch}} \times \text{volume of tire} = \text{volume of gas in tire}$$

So the pressure of the carbon monoxide in the tire, divided by the normal atmospheric pressure of 15 lbs. per square inch, times the volume of the tire is the volume of the carbon monoxide gas in the tire.

Right around the tire change time, the rate of bubbling may slow up again. This means it is time to add another dose of sulfuric acid to the mixture. Put another 100 ml of concentrated sulfuric acid into the separatory funnel, and let it flow into the 3-necked flask. This will get things going again, especially after it gets swirled to mix it in.

So now that our determined poisoner has this supply of conveniently bottled carbon monoxide, what does he do with it? He moves to the attack, and delivers it to the mark. The easiest way for him to do this is to drive up to the target with the carbon monoxide filled tires in the trunk, roll them up to the dwelling to be gassed, and let the carbon monoxide out of them through a section of tubing into the dwelling. A small nick taken out of the corner of a window works fine for introducing the gas into the target. Since carbon monoxide is very close to the same weight as air, it will mix evenly with all the air in the target eventually. A nice touch to help cover tracks after the job is to obstruct the furnace exhaust before leaving the scene.

A useful variation on this scheme is for the victim's car to be attacked instead of his home. This has the advantage that the car is often easier to get to than a man's home. Locked doors are easily opened with a thin strip of metal, and the gas piped in before closing the car back up. This works best when the target is going to be taking a fairly long trip with the car, so that such a large amount of carbon monoxide need not be used that it causes suspicion. The resulting traffic crash is likely to be fatal, even if the gas isn't. The

question which must be answered before embarking on this plan is whether the elimination of the victim is so important that it is worth exposing innocent people to risk when the target becomes drowsy on the road.

Carbon Dioxide

Carbon dioxide (CO_2) is not usually thought of as a deadly poison by the general public, but it is, in my opinion, one of the best of all poisons. The autopsy on a person who had been poisoned with carbon dioxide would show nothing, just that the victim had stopped breathing and had suffocated. Not the forcible suffocation with a pillow or whatever that is seen so often in old movies. No, that is so obvious because a person who is being strangled or suffocated will vomit up into his lungs in a vain attempt to clear his airways. He will also inhale fibers of whatever is being used to suffocate him. All this can be found quite easily at autopsy. Instead, the person who breathes in too much CO_2 actually drowns in the gas. Upon examination of the body, it will appear that the victim stopped breathing in his sleep. This is not that rare an occurrence. People who suffer from a condition called apnea will often die in their sleep because they forgot to keep breathing, or the lack of air can cause a heart attack. People who have apnea usually do not know that they stop breathing in their sleep, unless their bed partner tells them of the condition. It is a common cause of early and mysterious death. An attack with carbon dioxide can easily look like death from apnea.

CO_2 has two main drawbacks to its use as a poison. First of all, a large amount of it must be used to cause lethal effects. The generally accepted lethal dose of carbon dioxide is 4%, over a fairly long period of time. To be sure of the job, the attacker would want to double this level to 8% CO_2. This is a large amount of gas, especially if a large area is to be gassed. The large amount which must be used also makes it difficult for all of the gas to dissipate

out of the attack area before the body is discovered. A finding of "died in his sleep of natural causes" is not to be expected when the people who discover the body keel over shortly after entering the room.

Secondly, only a sleeping person can be easily attacked with CO_2. A person who is awake would notice the headache, dizziness, and rapid breathing caused by carbon dioxide. As a consequence of these two drawbacks, it may be difficult or impossible to mount an attack with CO_2 on a target who sleeps holed up in a heavily fortified Fuehrer-bunker.

CO_2 is probably the most easily available deadly substance. Its two most common forms are as dry ice and as a pressurized gas in cylinders. The cylinders are most commonly seen in welding shops, and hooked up to beer kegs to keep them from going flat once they have been tapped. Many liquor stores carry these cylinders for their keg beer customers.

One pound of CO_2, in either the form of dry ice or inside a cylinder, will, when expanded into the air, take up a volume of a little over 8 cubic feet. So to calculate the amount of CO_2 needed to produce a deadly concentration of CO_2 in a room, we just find the volume of the room, and divide it by 10 to get how much CO_2 is needed to produce a 10% concentration of CO_2 in the target.

For example, if a room is 15 by 15 by 8 feet, the volume of the room is 1800 cubic feet. One tenth of that is 180 cubic feet. Since one pound of CO_2 is about 8 cubic feet, we divide 180 by 8 and get the answer of 22.5 pounds of CO_2 needed to produce a 10% concentration of CO_2 in the room. In dealing with cylinders, it is vitally important that pounds of pressure showing on a gauge not be mistaken for how many pounds of CO_2 are in the cylinder.

Chapter Three
WAR GASES

The scene is France, 1915, and the armies of Europe are locked in bloody, stalemated trench warfare. In an effort to break this deadlock, the German Army called upon the most powerful segment of its economy, the chemical industry and the brilliant chemists behind it, to give them a weapon for victory. Thus was born the modern era of chemical warfare.

The first chemical used was chlorine. The results on the unprotected Allied soldiers were spectacular. Thousands of soldiers were either killed or seriously injured in one attack. Chlorine, however, has a serious drawback. It is very unpleasant to breathe, and so gives a warning to the people being gassed. It is, in fact, pretty nasty at concentrations much too low to cause injury over a short period of time. The only way people could be gassed with chlorine is if they could not escape from the cloud of gas. You can prove this to yourself if you want, by mixing a small amount of bleach and Saru-Flush in a toilet bowl, just before flushing it down and escaping the area. The powerful bite of chlorine is impossible to ignore.

Phosgene

To overcome this problem, a new and much more powerful gas was introduced, phosgene. Phosgene ended up causing more death and injury than any other gas used in World War I. It was very

sneaky, in that people often did not realize they were being gassed with phosgene, and its effects were often delayed for a day or two. A few stories from World War I will help to illustrate these points.

One day during the war, a British sergeant was working on some emptied cylinders of phosgene, disconnecting some pipes from them after an attack on the German lines. Upon taking apart one of the pipes, he got a small whiff of phosgene. He didn't think anything of it, and just carried on with his work. He got a good night's sleep, had a fine breakfast, but soon after, he became very sick and died 24 hours after his fatal whiff. Moral of the story: people generally don't realize that they are being gassed with phosgene. It is subtle, but devastating.

One more story: One day, after a British phosgene attack on the German lines, a captured German soldier was brought in for questioning. In great spirits, he scorned the British gas as useless. The next day he was dead. Moral of the story: people *like* to be gassed with phosgene. The euphoria that the German soldier felt is a pretty typical symptom of phosgene.

As you can see from the above stories, phosgene was a great advance for the art of poisoning. It is equally useful for the assassination of one individual, or for assaulting heavily fortified installations. For example, an ingenious soldier of fortune could topple a banana republic single-handedly by introducing a little of this gas into the ventilation system of whatever building the ruling junta happens to be meeting in. They would not even realize they are under attack, until they keel over the next day. Many other strongholds of the power system (yes, *those* places!) are equally vulnerable to the kind of insidious attack which is possible with phosgene, or one of the other war gases described in this book. Why this idea has not been put into practice before, I cannot imagine. It speaks poorly for the quality of mercenary fighting going on these days, too much conventional thinking.

Now that you see how potentially useful phosgene is, let's get down to the nitty-gritty of what it is and how to get hold of some. Phosgene is also called carbonyl chloride, or carbon oxychloride. It

has the chemical formula $COCl_2$. It is a gas at normal room temperature because it boils at about 8° C, but with a little extra pressure, it becomes a liquid at room temperature. For this reason, it is often stored and shipped as a liquid in cylinders, or in railroad tank cars. It is not hard to imagine the havoc which would be wreaked if one of these tank cars blew up while passing through a populated area. Phosgene has a faint smell which has been described as like musty hay or green corn.

Phosgene is very poisonous, but just how poisonous it is has not been agreed upon. Depending on which source you believe, the fatal dose of Phosgene for a 30 minute exposure is anywhere from one part in a million air to one part in 50,000 air. The figure I put the most faith in says one part in 100,000 air for a 30 minute exposure.

Phosgene has some quirks which must be taken into account to effectively use it. First of all, it is quite a bit heavier than air, so it will sink. It would not be very effective to just pipe some of it into a room, and expect it to make it around the target area from there. Introducing it into the ventilation system would be much more effective. The necessity of introducing it into the ventilation system could be avoided if fans or other active methods of keeping the air moving in the target building are in use.

Phosgene also has a strange effect on the taste of tobacco smoke. It makes what would normally be a fine smoke taste like rolled up hay. Whether this would alert the mark(s), or just make them curse their brand of smokes is difficult to say, but this property of phosgene should be kept in mind. The attackers could use this to their advantage to warn them when they are being exposed to phosgene. The change in tobacco taste is especially pronounced in pipes.

Phosgene has another strange property in that it will rust metal objects when it gets out in the air. This is because Phosgene reacts with the water in the air to make hydrochloric acid, which then rusts metal. This rust takes a little while to develop, so it will not alert the victims, but it could be of help to investigators later, to determine the site of the attack. This reaction of phosgene with water makes phosgene useless in rainy weather.

The medical effects of phosgene are very interesting. At the recommended concentration, about one part in 100,000 air, the victim does not notice anything. Perhaps a slight tingle in the throat or smarting in the eyes, but this very quickly passes and all seems well to the mark. Phosgene gas has no color, so there is nothing he would see which would tip him off. Following the exposure to the gas, the victim feels fine. He may, in fact, feel euphoric. Little does the victim know that his lungs have been seared, and are filling up with fluid, while he is feeling so good. Then, unfortunately for the target, he begins to feel sick. Just how long it takes to begin feeling sick depends on how large a dose of the gas the victim received. With smaller doses, it could take as long as 2 days.

The victim finds that the most insignificant effort will send his pulse through the roof, and his tortured lungs battling for breath. The person literally begins to drown in fluid seeping from his lung tissues. It comes up to the mouth as a watery fluid with some blood in it. The victim may gurgle up a half a gallon of this stuff per hour, until he finally loses the strength to cough it up. This could take as long as two days to do.

Naturally, a person dying this way is probably not going to be listed as a natural death. The question is, what will it be listed as? People with failing hearts often die with their lungs filled with fluid, as do heroin junkies suffering from overdose, but their lungs are not seared like the phosgene victim. Unless the victim liked to sniff glue and other strange things, it is a good bet that an investigation would be launched after death to determine whether the victim accidentally came into contact with something which seared the surface of his lungs, or was murdered. Because this is such an exotic way to die, odds are going to favor the finding of accidental death, unless some strong bit of evidence shows up (i.e., witnesses, insurance or other motive, or finding the phosgene-rusted gas site) to show that the victim was murdered. Of course, purchases of phosgene by a suspect would be damning evidence.

How then does a person get some phosgene? For some, it may be simplest to steal some or to have it given to them from a connection

in the chemical industry who can heist it for them. Phosgene is used a lot in the dye industry, and to a lesser extent throughout the chemical manufacturing industry. It is usually found in gas cylinders under pressure, although it is sometimes found as a solution in toluene.

For those of us who don't have the connections to get phosgene for us, it is wise to make some up from scratch. It is not particularly hard, nor dangerous, if some basic precautions are taken. As always, take care, work while sober, and safety first!

Phosgene was first made in 1812 by John Davy by mixing together carbon monoxide and chlorine, and letting the mixture sit in the sunlight. This is how phosgene got its name, "phos" meaning light and "gene" meaning made from. This is an interesting reaction, but it is just not practical for cooking some up because the amount of product made is so low. You mostly get back the carbon monoxide and chlorine you started with; only a small amount of phosgene is made.

The giant chemical manufacturers make phosgene by passing a mixture of chlorine and carbon monoxide over a specially made catalyst of charcoal. This is a good, cheap method, but it is not suitable for the home experimenter.

The Poor Man's James Bond claims that phosgene can be made by setting a pan filled with carbon tetrachloride (dry cleaning fluid) on a radiator and letting it sit. Sorry about this, Kurt, but it just ain't so. The carbon tetrachloride will just heat up until its boiling point of 76° C is reached, then it will just boil away. No measurable amounts of phosgene can be made by this method. Perhaps this misunderstanding got started because if carbon tetrachloride is splashed onto *red hot* metal, a fair amount of phosgene is made. This is one of the dangers of using a fire extinguisher charged with carbon tetrachloride, and could perhaps be used in an emergency to make some phosgene. You can bet that you will never see that one on *MacGyver!*

The best way for phosgene to be made on a fairly small scale is by reacting fuming sulfuric acid with carbon tetrachloride. Carbon tetrachloride is fairly easily available at the hardware store at a very

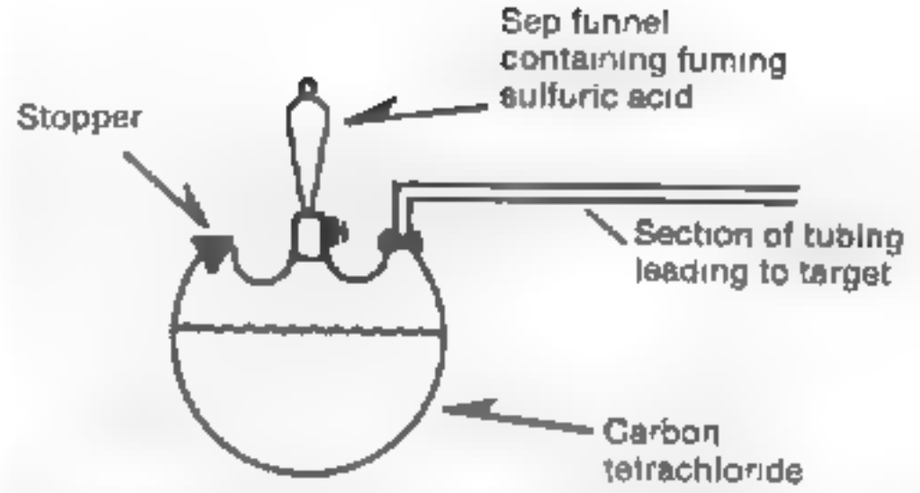
cheap price. It is becoming scarcer lately because it has been found that prolonged exposure to carbon tetrachloride can cause liver problems, but it can still be found at these easy sources. Fuming sulfuric acid is a fairly common laboratory chemical, costing about $35 per pint when purchased in small amounts. It is a mixture of sulfur trioxide in sulfuric acid, with the usual concentration of sulfur trioxide (SO_3, also called oleum) in the sulfuric acid being in the range of 20%

The reaction that actually takes place is between the sulfur trioxide in the fuming sulfuric acid, and the carbon tetrachloride.

$$\underset{\text{sulfur trioxide}}{SO_3} + \underset{\text{carbon tetrachloride}}{CCl_4} \longrightarrow \underset{\text{phosgene}}{COCl_2} + \underset{\text{chloro-sulfonic acid}}{2SO_3HCl}$$

Phosgene is produced, and bubbles out of the mixture. The chlorosulfonic acid produced as a byproduct stays as a liquid dissolved in the unreacted sulfuric acid. No heating is needed to make this reaction go, so production of phosgene is very quick and simple. Grenades producing phosgene can also be devised.

To make phosgene, the same combination of separatory funnel and 3-necked flask is used as was shown in the section on carbon monoxide. Instead of the tire storage system used for carbon monoxide, however, we have instead used a length of vinyl tubing leading to the target. This is because phosgene does unbelievable things to rubber, like turning it into an oily goo. For this reason, the phosgene must be directly pumped into the ventilation system of whatever building is to be attacked. The air intake of an air conditioner is a very convenient place to introduce gases into a building. The draft that the conditioner makes will help to keep the phosgene stirred up in the air, rather than letting it settle into the low points of the building.



A convenient sized batch of phosgene to show how this system works is about $^2/_3$ of a cubic foot of phosgene. This is enough phosgene to make a one part in 50,000 concentration of phosgene in a building 33,000 cubic feet large. In calculating the amount of gas actually delivered to the target, it must be remembered that the gas first has to fill up the flask and the length of tubing. So the volume of the air space left above the chemicals in the flask, and the volume of the inside of the length of tubing must be subtracted from the total amount of gas produced.

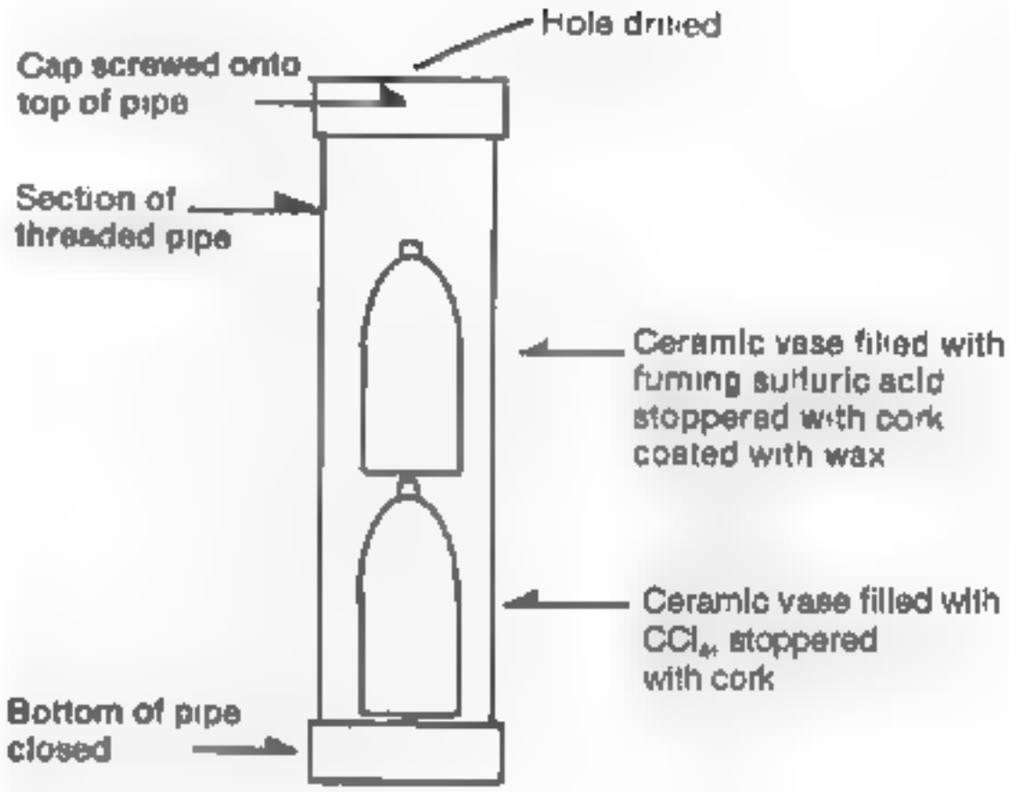
Now to begin. To make $^2/_3$ cubic foot of phosgene, 100 ml of carbon tetrachloride is put into the 3-necked flask. The section of tubing, stopper, and separatory funnel are all put securely in place. Then 230 ml of fuming sulfuric acid is put in the separatory funnel. (Careful! Fuming sulfuric acid is even worse than regular sulfuric acid. Always wear gloves and eye protection.) The valve of the separatory funnel is opened a little, and a slow stream of fuming sulfuric acid is allowed to flow into the 3-necked flask. It is swirled around to help mix it in, and almost immediately phosgene begins to bubble out of the mixture. The rate of addition of fuming sulfuric acid should be controlled to keep the amount of phosgene bubbling out at any time under control. The reaction is pretty well done about 5 minutes after the last of the fuming sulfuric acid has been added to the carbon tetrachloride, so long as the mixing was OK. The liquid left in the flask can then be rinsed out with water (watch out for more fumes!) and the whole system packed up and moved out of the area.

Now for a few notes on this process. The amount of fuming sulfuric acid used was calculated on having the 20% sulfur trioxide type of fuming sulfuric acid. If the fuming sulfuric acid was instead 40% sulfur trioxide, then only half as much would have to be used. There is also no need to stick to the $^2/_3$ cubic foot sized batch. A batch of any size can be made. To make half as much, use half as much of both chemicals. Finally, it is vital that only fuming sulfuric acid be used in this reaction. If ordinary concentrated sulfuric acid is used, the reaction takes another course. Hydrogen chloride gas (HCl) is produced along with the phosgene, and this is very unsatisfactory because the intense bite of HCl when breathed in would alert the victims. The reaction done this way also requires heating, and the addition of infusorial earth as a catalyst. (See *Chemical Abstracts*, Vol. 17, page 1395, and Vol. 13, page 2492.)

A grenade for phosgene would be a very handy thing. The attacker(s) could then just throw it at the target and take off, instead of having to stick around to supervise the delivery of phosgene to the target. The commotion caused by throwing a grenade will probably ruin the element of surprise and stealth for the attacker, but there are situations where this is justified. For example, a grenade dropped down a large ventilation shaft would probably never be noticed until it was too late.

In designing a grenade for phosgene, we come to several difficulties. First of all, both ingredients used in the making of phosgene are liquids. This makes the design of a grenade more complicated than if one of the ingredients was a solid. Secondly, fuming sulfuric acid is a really nasty substance. It gives off fumes that develop pressure if it is warm. It is also really corrosive to a wide variety of substances. To complicate things further, the two ingredients must be held together in one place long enough for them to mix and start to react.

The design you see on the next page solves these difficulties, and works reasonably well.



The idea here is for the fragile ceramic vases to be shattered when the grenade hits paydirt, allowing the chemicals to mix, spewing phosgene out the hole drilled in the top of the pipe. For this to work every time, the containers inside the pipe must have some room to fly around inside the pipe so that they are sure to be shattered on impact. The cork in the container of fuming sulfuric acid must be coated with wax to keep the acid from dissolving the cork while the grenade is in storage. As an alternative, the CCl_4 could just be poured into the pipe instead of kept in a container in the vase. This would help shrink the size of the grenade for easier throwing. The disadvantages of doing this are: (1) the grenade would have to be kept upright to keep from spilling the CCl_4, and (2) the pool of liquid could act as a cushion for

the vase containing the fuming sulfuric acid, preventing it from breaking on impact. This design could be scaled up for use as a bomb to be dropped from airplanes. The grenade should contain at least twice as much fuming sulfuric acid as carbon tetrachloride, just as with the gas generator.

Arsine

Arsine (AsH_3, also called arseniuretted hydrogen) is a very good general purpose poison. That's not to say that it is a perfect poison. No, it leaves traces which could conceivably be detected. These traces are very slight, however, so they may not be connected to the sorry condition of the mark. The trace that arsine leaves is a small trace of arsenic in the urine. Whether this would be enough to allow the coroner to piece together the clues and diagnose arsine would depend upon the skill of both the coroner and the poisoner. After all, we all have some arsenic in our bodies, along with traces of every other element known. It becomes a matter of judgment to say at what point the level of a substance in the body becomes abnormal.

Now that you've heard the bad stuff about arsine, let's move on to its good points. It is a very deadly gas. A concentration of one part in 100,000 over a few hours is deadly. Over shorter periods of time, the exact lethal dose is a matter of debate. The figures given here are from *The Toxicology of Industrial Inorganic Poisons*, which states that one part in 2000 air over a few minutes is deadly. Over a period of about half an hour, one part in 4000 is deadly. Over a period of an hour, one part in 60,000 can cause death.

Arsine is also very exotic. This is an important point in its favor, because it means that the correct solution is much less likely to come to the minds of the people doing any post-mortem investigation. Poisoning by arsine is occasionally seen in industrial accidents, but not often enough to keep it at the forefront of the minds of coroners. I have never heard of a case of homicidal poisoning with arsine. That alone is enough to recommend it.

The symptoms of arsine poisoning do not help much in diagnosing it. To start with, it is not irritating to the subject while he is being dosed with the gas. At the high concentrations needed for lethal effect over a few minutes time, there is an awful smell like garlic, but at the lower concentrations it is nothing that would cause alarm or stick out in his memory. After being dosed, there is a latent period of up to a day or so before any symptoms show up.

When the first symptoms appear, they don't mean very much. The subject feels lousy, has trouble breathing, vomits a lot, and has blood in the urine because his red blood cells are being dissolved. Eventually, his kidneys shut down because they get plugged up with debris from those broken up red blood cells. This breakdown of the red blood cells is called hemolysis, and is a symptom of a wide variety of conditions.

Finally, arsine is a very easy poison to make and use. The ingredients are cheap and plentiful (except for arsenic trioxide, As_2O_3, which would be most wisely heisted) and safe to handle until they are mixed together to make arsine. A grenade for arsine is much simpler and more productive than the phosgene grenade.

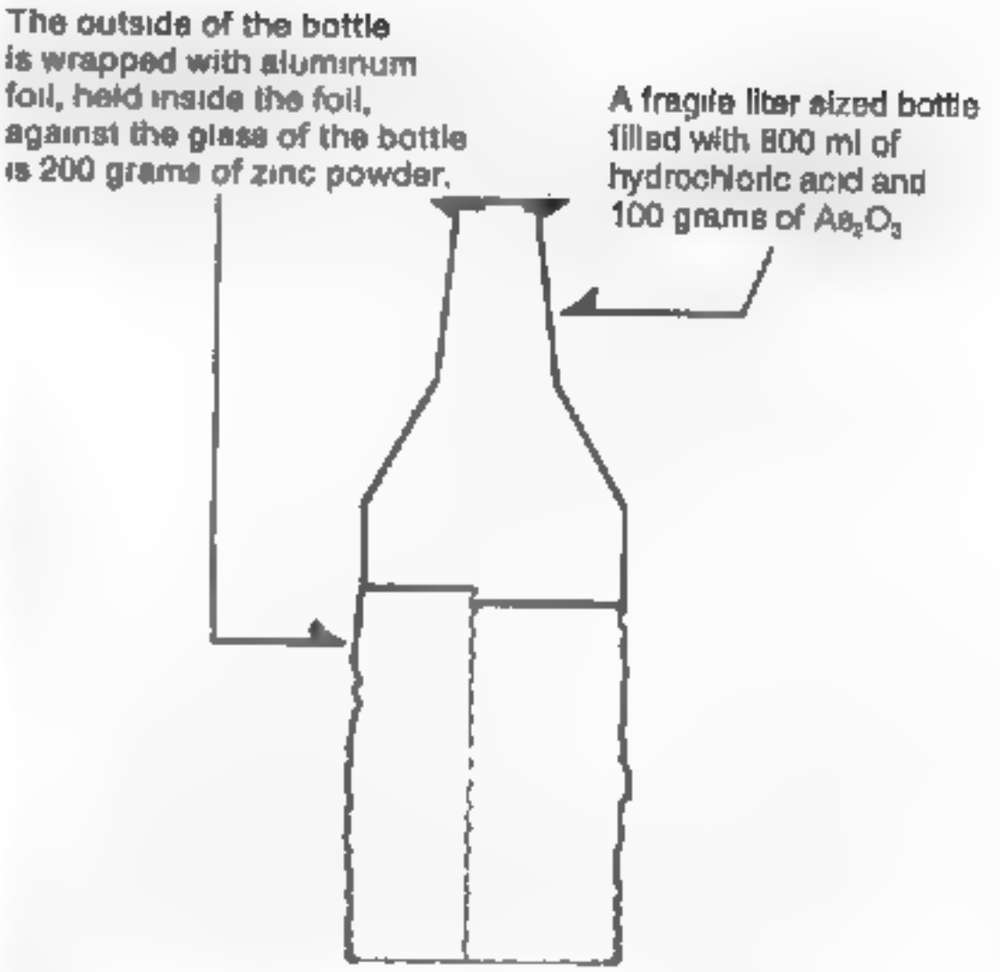
There are two really good methods for making arsine. One is about as good as the other, and the method chosen depends on which raw materials the attacker can most easily get his hands on. Method number one is based upon the reaction that takes place between zinc powder and hydrochloric acid. Many school boys have found out that when you mix these two together, the zinc fizzes like crazy, producing hydrogen gas. While zinc is undergoing this reaction, it becomes what is termed a powerful reducer. If some arsenic trioxide is mixed into the brew, it will get reduced, which in this case means its oxygen atoms get replaced with hydrogen atoms, and arsine is produced along with whatever hydrogen doesn't get used up.

$$\underset{\text{zinc}}{3Zn} + \underset{\text{hydrochloric acid}}{6HCl} + \underset{\text{arsenic trioxide}}{As_2O_3} \longrightarrow \underset{\text{arsine}}{2AsH_3} + 3ZnCl_2$$

This is nowhere near so complicated as it may seem. In fact, actually doing it is so simple that I'm sure a monkey could be trained to do it. To get going making some arsine, the same type generator used to make carbon monoxide and phosgene is used. (And you didn't believe me when I said no home is complete without one?) Into the flask part of the set-up is placed 30 grams of arsenic trioxide and 200 grams of zinc powder. These two ingredients should be mixed together as thoroughly as possible before putting them in the flask. Then another 70 grams of arsenic trioxide is weighed out and it is dissolved in hydrochloric acid. The hydrochloric acid I would recommend for this purpose can be obtained from the local hardware store and is 28% hydrochloric acid. It may be sold under the name muriatic acid. Seven hundred (700) ml of it will be needed, so the attacker will measure out that much and put the 70 grams of As_2O_3 in it. The acid-arsenic mixture is then poured into the separatory funnel, and when the attacker is ready to make arsine, the valve on the separatory funnel is opened, and the mixture is allowed to run down onto the zinc powder in the flask.

When the acid hits the zinc, gas is produced like crazy. Some of it is hydrogen, and some of it is arsine. It can be piped into the target with a section of tubing, or it can be stored in an automobile tire. Because of the extremely poisonous nature of arsine, this may only be attempted under the best conditions of ventilation. The poisoner could all too easily become the poisonee trying to fill a tire with arsine without excellent ventilation. The amount of arsine produced according to these directions is about $^2/_3$ cubic foot.

A grenade for arsine using these chemicals is pretty easy to put together, and could be a lot of fun. See the diagram on the next page. When the bottle breaks, the chemicals mix together, and a cloud of arsine is produced. The results can be devastating in an enclosed area. One drawback that arsine has as an assault poison is that it does not have good knock-down power. Its effects are delayed for a day or so. A wise tactician will keep this in mind when planning an assault.



The other good method for making arsine is by reacting diluted sulfuric acid with zinc arsenide (Zn_3As_2). Zinc arsenide may be difficult to find, but it has the advantage that arsine is simpler to make with it. To make 1½ cubic feet of arsine, 350 grams of Zn_3As_2 is placed in the flask, and then about a quart of battery acid (available at hardware stores and garages) is run down onto it from a separatory funnel with swirling of the flask to mix them together. A grenade using these chemicals would have the bottle filled with the battery acid, the bottle would then be wrapped with foil, and the space between the bottle and the foil would be filled with 350 grams of Zn_3As_2.

Now for a last few words on arsine. Arsine is a flammable gas, so it would catch on fire if a stream of it was spewed into an ignition source. It is also quite a bit heavier than air, so it will settle into whatever low areas it can find, unless the air in the target area is kept moving with a fan or something similar. For more information on arsine and phosgene, see *The Medical Aspects of Chemical Warfare* by Edward Vedder. This fine book gets into real detail on just what these chemical warfare agents will do to the human body, and also goes into some detail on the (futile in the case of arsine) medical treatments used for the victims of theses gases.

Phosphine

In a little known gas named phosphine, we encounter what is, without a doubt, the very best gas for producing "death by causes unknown." This ability that phosphine has for evading the coroner's attempts to find the cause of the mark's demise is a strangely under-reported, yet exciting ability. Those "in the know" seem to think that it would be reckless and irresponsible to let the public know just what are the limits of their abilities. For them it is much better to let the public believe in their infallibility and omniscience. This is typical of the way that the power structure in this supposedly free country manipulates the flow of information to the public. To their credit, you have to admit that they do a really masterful job of manipulating the media and the resulting flow of information, since the vast majority of the population, and even the media swine themselves do not notice the blinders they wear. After all, propaganda is what they get in Russia. Here we get the "news."

Phosphine's ability to disappear in the body can be explained pretty simply. The chemical formula for phosphine is PH_3, meaning it is composed of one atom of phosphorous, and three atoms of hydrogen. As I'm sure all of you know, your body has a lot of phosphorous floating around in it, for building teeth and bones, and to perform a wide variety of other functions in the cells. Most of this

phosphorous is in the form of phosphate (PO_4) and pyrophosphate (P_2O_7). When phosphine is absorbed into the body, it first does its dirty work, producing symptoms similar to arsine, then it gets oxidized to the forms normally seen in the body. For this reason, when Quincy and his ghoulish brothers at the morgue start examining the bodily fluids of a victim of phosphine, there is nothing for them to see. Phosphine certainly comes close to perfection as a poison!

Perfection, of course, has its price. That is the way of the world. In the case of phosphine, the price is that it is difficult to make, deliver, or store safely. That is not to say that the task is impossible. Quite the contrary. It is well within the ability of almost everyone to use phosphine effectively. It just must be understood up front that phosphine requires more care in its use than any other substance discussed in this book, with the exception of the nerve gases.

The cause of this difficulty is the fact that phosphine, as normally prepared, will burst into flames when it comes in contact with air. This destroys the phosphine, making the attack futile.

The resulting flames could also injure the would-be attacker, and spoil any element of surprise the attacker may have had going for him.

Phosphine itself does not spontaneously burst into flames in air, but an unavoidable byproduct of making it, diphosphine (P_2H_4) does, and is the spark that sets off the whole mixture. The method given here for producing phosphine overcomes this difficulty, allowing the diphosphine to do its unavoidable thing without setting off the whole mixture. If a resourceful poisoner could obtain a cylinder of pure phosphine from a laboratory somewhere, all this hassle could be avoided and the gas could be used directly from the cylinder. These cylinders are hard to come by, however, because phosphine is lacking in practical uses in the laboratory. It is some pretty exotic stuff, so exotic that the majority of chemists have no idea the stuff exists. It also ranks pretty close to the last thought that would enter the mind of the typical coroner.

Phosphine is a very deadly gas, about as deadly as arsine. It is colorless and has a foul smell that has been compared to rotten fish.

This foul smell means that it must be used at the lower concentration levels to keep the target from being driven from the poison zone by the odor. It may also be useful to mask the smell with heavy doses of room freshener or whatever else may be useful for covering the smell of rotten fish.

The deadly dosage of phosphine is about the same as for arsine. As usual for gases, the deadly concentration depends on how long the victim will be breathing the mixture. In the case of phosphine, it is quite acceptable to go well over the lethal limit to be sure that an exceptionally tough mark does not escape his fate. This is because phosphine is destroyed in the body, so there is no need to keep doses to a minimum to make its detection more difficult. In my opinion, the best dosage of phosphine is a concentration of one part in 4000 for a period of at least an hour. Lower concentrations of it can be used if the exposure is longer.

The symptoms of phosphine poisoning take a while to come on, especially at the lower doses and concentrations. Up to a day or two can pass before the symptoms begin, so it is a very sneaky poison. The symptoms start with a difficulty in breathing along with a pain in the chest. This leads into the rest of the symptoms of arsine poisoning, leading finally to death if a fatal dose has been absorbed, because there is no antidote for phosphine poisoning.

There are two pretty good methods for making phosphine. Both of them are gas generator type processes. There is no grenade for phosphine because of its tendency to burst into flames on contact with air unless precautions are taken.

The method of making phosphine that uses the simplest and most easily available chemicals is by reacting white (or it may be called yellow) phosphorous with sodium hydroxide (potassium hydroxide will work as well) in a solution of water. The reaction goes this way:

P_4 +	3NaOH	+ 3 H_2O ⟶	PH_3 +	$3NaH_2PO_2$
Phos-phorus	sodium hydroxide	water	phosphine	sodium hypo-phosphite

So yellow (or white, but not red!) phosphorous, which is in the form of P_4, reacts with sodium hydroxide in water to make phosphine and sodium hypophosphite, which is a solid that stays dissolved in the water. The byproduct diphosphine, which is the cause of its flammability in air, is not shown in the above reaction, but it is made nonetheless.

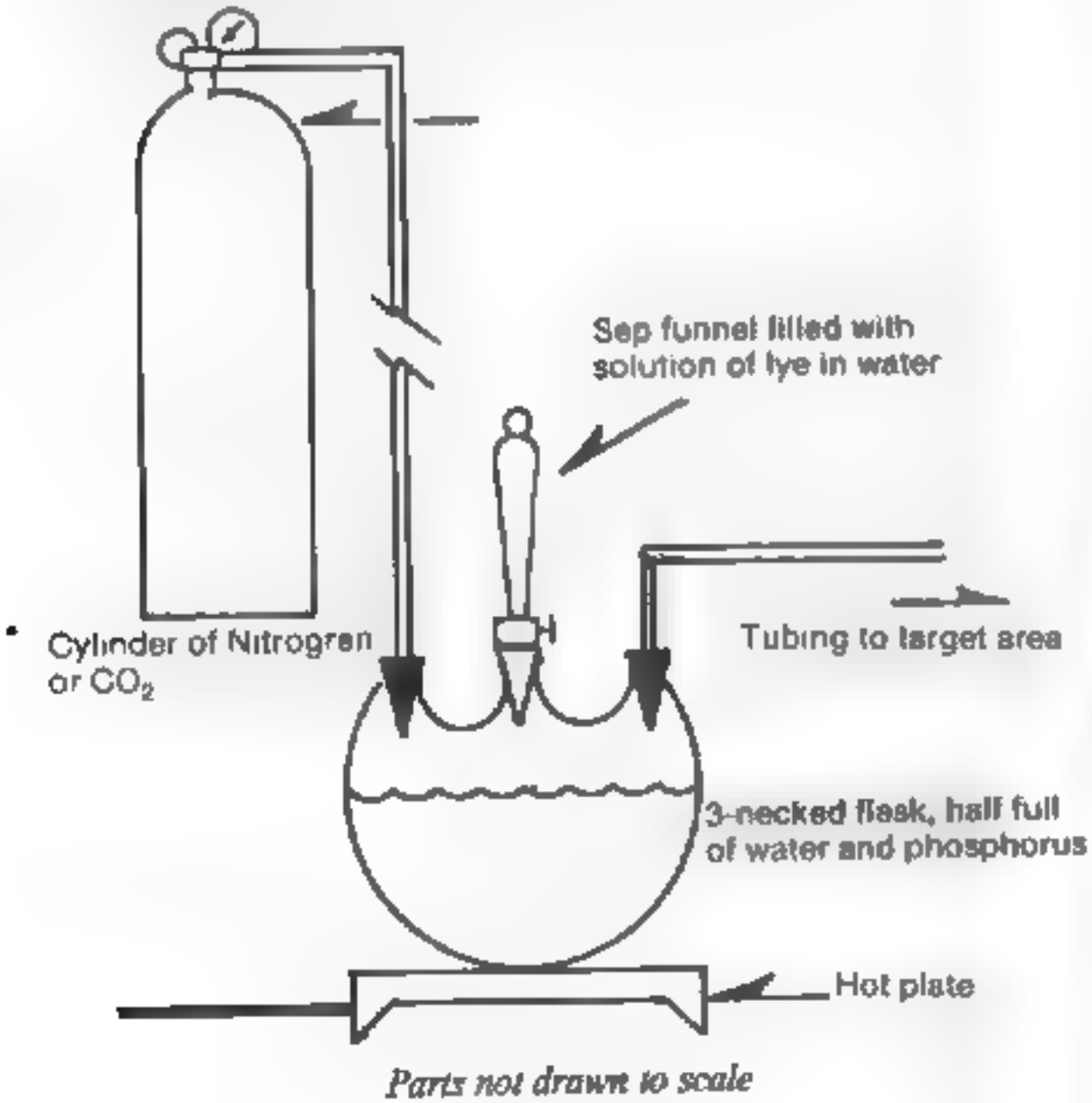
Now for a word about phosphorous. There are two common forms of phosphorous: red and yellow, which is often called white. The red form is not very dangerous and is used a lot in match manufacture. It is of no use in making phosphine. The yellow form is pretty dangerous for several reasons. First of all, it must be kept under water, or at least covered with a film of water, because it bursts into flames on contact with air. That explains its use in incendiary shells and bombs and fireworks. Secondly, it must not be touched with bare fingers, or nasty burns will result. Finally, it is a pretty good, but quite detectable poison when swallowed. About 50 milligrams will do a man in.

Now with all the preliminaries taken care of, here is how an attacker can make phosphine and deliver it to the target.

On the next page, we see a cylinder of nitrogen or CO_2 gas, which can be picked up very easily at a welding supply shop, attached to one neck of the now very familiar 3-necked flask. As usual, the center neck holds the separatory funnel, and the last neck is attached to a section of plastic (clear vinyl) tubing which leads to the target area to be gassed. The 3-necked flask is sitting on the coils of an electric hotplate.

When it is time to make phosphine, the attacker secures the 3-necked flask in an upright position and fills it nearly half full of distilled water. Then the sticks of yellow phosphorous are quickly put into the flask and submerged under the water. This must be done quickly so that the sticks of phosphorous don't dry off and ignite during the transfer. To make ¾ of a cubic foot of phosphine, 125 grams of phosphorous is required. Then 200 grams of a good quality lye such as Red Devil or High Test is dissolved in a pint of distilled water. After it is dissolved, a flow of gas from the cylinder is begun

and continued until the air is swept out of the tubing and flask. This will take a few minutes. After that the hot plate is turned onto the flask, and the water brought up to a simmer. Then the flow of gas is started up again, and the solution of lye in water is put into the separatory funnel, and it is run in 100 ml at a time at 5 minute intervals. During this time, about 30 cubic feet of gas should have flowed out of the cylinder, or a rate of 1 cubic foot per minute, if a flow meter is attached to the cylinder.



Parts not drawn to scale

This input of inert gas will dilute the phosphine down to about 2 or 3 percent of the total mixture. When it is this dilute, it is no longer able to be detonated when the diphosphine in it ignites on contact with air. In this way, phosphine can be made and used safely, with very satisfying results.

This same apparatus can be used to make phosphine from other chemicals. Exterminators may have a chemical called zinc phosphide, Zn_3P_2, for dealing with rats. It is a gray powder. If 260 grams of it is placed in the 3-necked flask (no water in the flask!) and a pint of hardware store hydrochloric acid is run down onto it from the separatory funnel, about 1½ cubic feet of phosphine will be made. Naturally, the flow of inert gas is required with this method also.

For more information on the effects of phosphine, see *The American Journal of Medical Science* Volume 208, page 223 (1944).

Chapter Four
NERVE GAS: THE POOR MAN'S ATOM BOMB

Ever since the detonation of the first atomic weapon in 1945, the top priority of each nation that came to possess these weapons has been to keep other "less responsible" nations from gaining access to these weapons, and above all to prevent their falling into the hands of private "terrorists." The very idea of some group putting together a "basement nuke" and dangling this weapon of decision over their heads is the ultimate nightmare for these "responsible" nuclear criminals.

All this concern is quite understandable, since nuclear weapons are extraordinarily spectacular and very macho. However, there is another weapon of mass destruction that is just as devastating to human life. A well-placed nerve gas bomb of sufficient size could inflict a death toll on a city comparable to that of a medium-sized nuke.

With all the scrambling that has taken place over the years by various groups and nations for the nuclear weapons of mass destruction, why has the chemical weapon of decision been so roundly ignored? I believe that a combination of factors are responsible for this situation. First of all, nerve gas is not loud and spectacular like a nuclear bomb is, so it does not inspire in the popular consciousness the perception of power and feelings of terror that a nuke does. Since the perception of power is just as important to the psychology of political struggles as actually

possessing it, this is a contributing factor towards the orphaning of these chemical weapons.

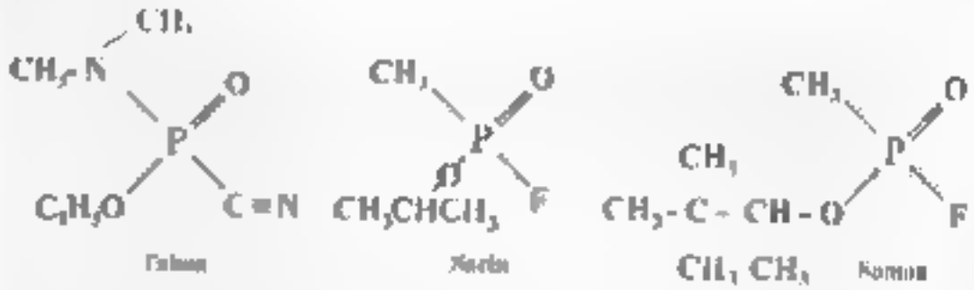
Secondly, nerve gas and other chemical weapons have not been popularized and romanticized by our culture the way that nukes have been. To illustrate my point, just consider the plethora of movies and books dealing with some aspect of nuclear devastation or its aftermath. This list is nearly endless. Add to this the many pressure groups whose sole purpose is to agitate for the dismantling of nuclear weapons and power plants. There is just no counterpart to this for chemical weapons, in spite of their amazing destructive abilities. The sole exception to this that I am aware of is a movie called *And Millions Will Die* starring Richard Baschart, made in 1973. This movie dealt with a nerve-gas bomb planted in Hong Kong, and the efforts of authorities to defuse it.

Finally, there is the almost universal belief that the only way any person or group could obtain a nerve-gas bomb is to steal one from an Army stockpile. The origin of this strange belief is pretty hard for me to fathom, but it has probably been planted in the popular consciousness by the government through their lackeys in the media. Theft may be the only way any group short of major governmental status could obtain a nuke, but this is definitely not the case for nerve gas. After all, nerve gas is just a chemical, and not a very complicated or difficult substance to make. The experience in modern times with clandestine drug laboratories should be enough to make clear my point that just because a chemical is not available through regular channels doesn't make it disappear, or even that hard to get.

What exactly is nerve gas? Well, to be brief about it, it's bug spray for people! It is closely related to those organophosphorus compounds used so widely as insecticides. It is also not a gas, but rather a liquid with a very high boiling point. To be used to gas an area, it must be dispersed into the air as an aerosol like out of a spray can. It can also be dispersed as a mist in the air by detonating an explosive charge in it. When done correctly, both methods work well, although the aerosol spray can has the obvious advantage of

stealth. This is not to say that the explosive method of dispersing it is without its merits. An explosion is always great for bringing out crowds of curious onlookers into the open, where they can easily fall victim to the effects of the gas.

Nerve gas is a generic term which covers several related substances. All of them have an atom of phosphorous at their heart, with a variety of other chemical groupings attached to the phosphorous atom, depending upon the particular nerve agent. The molecular structures of the three main types of nerve gas are shown below:



These compounds are shown in order of increasing deadly potency. Sarin is about 10 times more potent than Tabun, and Soman is about three times more powerful than Sarin. All of them are mindbogglingly deadly. An interesting feature of nerve gases is that they do not have to be breathed in to be deadly. The tiniest droplet on the skin will quickly work its way into the body and cause death. This tiny amount can easily be picked up by brushing against a contaminated object, so an area that has been nerve-gassed will be a death trap until the nerve gas has been broken down by water and the sun. Breakdown could take a couple weeks, depending on weather conditions. For this reason, military strategists think that nerve gas is the perfect weapon to guard the flanks of advancing armies against counterattack.

How do these nerve gases cause death? To explain this, one must first know a little bit about how nerves cause muscles to contract. Where the nerve connects to the muscle, there is a little junction

area. When the signal comes down the nerve to the muscle telling it to contract, the end of the nerve releases a little bit of a substance called acetylcholine into that junction area, and this signals the muscle to contract. As soon as the muscle obeys, an enzyme called cholinesterase destroys the acetylcholine so that the muscle can relax again and be ready for its next command. If the acetylcholine was not destroyed, every muscle in the body would soon be stuck in the "on" position and no movement would be possible, just writhing and twitching as muscle struggled against muscle, until death quickly arrived because the victim could not breathe.

When nerve gas enters the body, each and every molecule of it goes on a search and destroy mission, looking for cholinesterase. When they find each other, the nerve gas ties up the cholinesterase, making it permanently useless for servicing the muscles. Soon there is no cholinesterase left in the body, and a most disgusting death scene results.

A good way to get an idea of what death from nerve gas is like is to spray some flies with Raid. It works the same way on flies that nerve gas does on humans. First the twitching, then no useful motion at all is possible. If people had wings, they would die on their backs, buzzing around in little circles.

Actually, the first sign of nerve gas poisoning is that the pupils of the eyes contract to tiny points, and everything becomes black to the victim. This early warning sign is very important to people working with nerve gas because it may be all the warning they get. From there the victim soon begins to twitch and collapse follows. The victim loses control of his or her bowels and is unable to breathe. Death follows in minutes.

Not all people are equally sensitive to nerve gas. It was discovered in the German nerve gas plants in WWII that fat people are better able to withstand the effects of low dosages of nerve gas. They also found that bathing in bicarbonate of soda (Arm and Hammer) solutions or ammonia is a good first aid procedure for minor spills on the skin. Injections of atropine is an antidote for mild poisoning with nerve gas.

Use of Nerve Gas

How best to use nerve gas depends upon the exact job it is called upon to do. Nerve gas is surprisingly versatile stuff, and can do any job from individual assassination to devastating large cities. It can also form an almost impenetrable barrier or make large areas uninhabitable for extended periods.

The use of nerve gas in assassination is its simplest use. This is because it does not present big obstacles in the logistics of the operation. Large amounts are not needed, and the area that must be contaminated can be ridiculously small. For example, some object that the mark is bound to touch over the next few days is laced with a droplet of the nerve gas. When the mark touches it, the nerve gas soaks through his skin, and death quickly follows. Examples of such an object would include watchbands, auto steering wheels, and doorknobs.

Would such a death remain a mystery? That is difficult to say. There is a test which measures the cholinesterase activity in the body of the deceased, and this test would quickly spot that something was wrong with a victim of nerve gas. This test is routinely done in some areas, and not at all in others. As a rule, areas where there is a lot of accidental poisoning with insecticides will do this test. Will they then be able to go and identify nerve gas as the cause of the poisoning? Probably not, so long as an excessive amount of it was not used, because it will all be tied up in the mark's body, bound to the cholinesterase. Nerve gas is not that easily analyzed with standard techniques even in its pure form because it breaks down under heat. So, the verdict is likely to be poisoning by substance unknown.

A nerve-gas attack designed to lay waste to a metropolitan area or to make some other strategic area uninhabitable would both be done basically the same way, so they will both be covered together

The simplest, and in several ways the best method for dispersing nerve agents over a population center is by a technique I call "blast

dispersion" for lack of a better name. It is the same principle that works in the nerve-gas artillery shell. A charge of explosive is used to heave the poison into the air and disperse it into a cloud of fine droplets which can then drift downwind over the target. There are some fine points to this technique which must be paid attention to for best results.

First of all, weather conditions are very important to the results obtained with a chemical attack. The best conditions are a nice summer day, with a steady wind of 10-20 mph. The nice summer-like conditions are important because under these conditions people will be out in the open, and the windows to their houses will be open, leaving them unprotected from the full effects of the nerve gas. The nice steady wind is important because it will help to move the cloud of nerve gas along so that it can spread its destruction over a wide area, rather than sitting in one place as it would on a calm day. Rain would be absolutely disastrous to the chemical attack because it would wash out the nerve-gas droplets prematurely, and also because nerve gas reacts with water, rendering it harmless. The amount of time required for this to happen depends on how acid the rain is, the more acid, the faster it goes. About 8 hours is required for rain of typical acidity. This reaction with water is the reason why nerve gas is not very useful for contaminating water supplies. Other aspects of the chemistry of nerve gases will be covered in the synthesis section of this chapter, but the property of reaction with water is so basic to an understanding of nerve gases that it belongs in everyone's general body of knowledge.

The second important fine point to the "blast dispersion" of nerve gases is that the cloud gets heaved up into the air, not splattered onto a bunch of walls in the neighborhood. Here the matter becomes a little more complicated, and there are several options open to the attacker to get the job done properly.

One option is to load the nerve gas into the warheads of a series of Roman candles, and let the blast of each one in the air disperse the nerve gas over the target. This suffers from the drawback that

each rocket can only carry a small amount of nerve gas. Add to that the erratic performance of roman candles and their limited range, and the fact that someone would have to make an easily traced sitting duck out of themselves to launch them, and you end up with an unsatisfactory solution. The last problem of having to attend the launch could be solved by tying the fuses for the rockets to a section of nichrome wire (toaster heating element) attached to a timer and plugged into the electric supply. The other problems with rockets are not so easily solved.

Another option is to place the nerve gas onto a rooftop, and set a charge underneath it to blast the gas skyward. This method is quite workable, but is filled with many potential pitfalls. For starters, the rooftops chosen should be at least 5 or 6 stories high, and should be the tallest buildings in the neighborhood. In this way the cloud of nerve agent will get lifted high enough into the air for a maximum spread, and will not get plastered onto the walls of a nearby building. It is essential that the charge of explosive be under the nerve gas, so that the blast lifts it upward, rather than driving it into a cement wall or floor. Each nerve gas bomb should have at least a couple gallons of active ingredient and sufficient explosive for this purpose is one that does not create a lot of heat in its explosion. The type of dynamite often used for mining purposes, called ammonia dynamite (monobel) is specially made to have a cool explosion. Blasting powder may also work.

The serious drawbacks to this method include the necessity of getting several high rooftops (only one would not result in a good coverage pattern of the target), installing a good-sized charge onto each one, and then maintaining security of each device from snooping busybodies, etc., until it is time for their detonation.

The third option is quite obviously my favorite. It is to attack with nerve gas the way it was meant to be used, via aerial assault. This could be most safely done using one or more ultralight aircraft as a bomber.

I really must digress here because this is a concept which has excited me for some time, and I feel its time is long overdue. The

ultralight is a nearly perfect aircraft for guerrilla or insurgent operations. It can be constructed cheaply and easily from common materials. Plans and kits for their construction are widely advertised in such magazines as *Popular Mechanics* and *Popular Science*. A plane so constructed is virtually untraceable since there is no paper trail of titles and serial numbers to identify it, should it be captured.

An ultralight needs no special airfield to take off and land in. A smooth stretch of dirt is all that is required to get a small air force going. Avoiding regular airfields means such paper shuffling nonsense as flight plans and radar beacons and registration numbers and pilot's licenses can be dispensed with. Deserted back roads also work admirably for take offs and landings as long as power lines are not strung in the way.

No great amount of skill is required to learn how to fly an ultralight. Lessons are available from local ultralights clubs and other, more conventional outlets. Maintenance of an ultralight is also pretty simple, since the typical engine comes out of a VW beetle.

The performance of a well-built ultralight is very satisfactory for its use as a bomber. Its typical cruising speed of 60 mph is slow enough for pinpoint accuracy on bombing runs, yet fast enough to outrun any ground pursuers. This is because the plane is not bound to follow roads, and so can take an escape course which autos can not follow. Upon completion of the mission, the plane could fly low and fast to lose contact with any pursuit, then land on a road in a wild area where the plane could either be abandoned (bad choice) or loaded up into the back of a truck.

The payload of a good ultralight can be heavy enough to do some real damage if the target and the weapons are well chosen. Up to 100 pounds can be carried on the plane, if the pilot is not too heavy. This amount carried in nerve-gas bombs would be enough to do some damage of historic proportions. Carrying any explosive more powerful than commercial dynamite (homemade nitro packs one

amazing punch), a target residential building can be vaporized by even a near miss.

It has long amazed me while reading and watching excerpts of gangland and other underworld battles, how much of the fighting is done by men on foot with peashooter-sized weapons. It is no wonder nothing ever gets settled in such a way. One can only wonder at the intelligence and imagination of these people when a weapon of decision can be constructed in a garage to obliterate the enemy right in his rat-hole.

The particular problem of delivering nerve-gas bombs via ultralight will now be discussed in detail. There are far fewer problems with this method than the others, so it gets my approval as best method. It is also the method most likely to bring a response of terror from the unaffected populations in other localities. The first two methods would undoubtedly remind them of Shiite Moslem tactics, and just rouse them to anger against their sand-dwelling enemies.

The design of the bombs is the first consideration of any attack. Since the bombs should explode in midair for best results, care must be taken in the construction of the detonation system. It is not just a matter of throwing them overboard and watching them splatter on the ground below. To ensure that the bombs explode in the air, several strict guidelines must be followed. First of all, the pilot must know at what altitude he is flying, and stick carefully to the planned altitude for the attack. A very satisfactory altitude for a nerve-gas attack is 1000 feet. This altitude is high enough for the safety of the pilot, yet low enough that errors in the height of bomb detonation will be kept to a minimum.

The actual design of a nerve-gas bomb designed to explode in midair should have the explosive charge in the center of the mass of nerve agent. Naturally, it would not do to have a stick of dynamite floating around in a jug of Sarin, and the active ingredient splashing all over the attackers. Instead, three 40-ounce beer bottles could be taped together in a triangular pattern, and a stick of dynamite

placed in the center space between the three bottles. The result is a bomb containing just short of one gallon of nerve agent.

Controlling the height at which the bomb goes off requires that the fuse attached to the explosive charge be of good quality, burning at a steady and predictable rate. The time required for an object to fall a given distance can be calculated using the following formula.

$$\text{Time to fall (in seconds)} = \sqrt{\frac{2 \times \text{the height to fall}}{32 \text{ feet per second}^2}}$$

So the time it takes an object to fall a given number of feet is the square root of twice the height of the fall divided by 32. For example, if the bomber is cruising along at 1000 feet, and detonation is desired at 150 feet, the bomb has 850 feet to fall. Twice 850 feet is 1700 feet. Seventeen hundred (1700) feet divided by 32 is 53. The square root of 53 is a little over 7.25 seconds. To get detonation at the height of 150 feet, a section of fuse taking about 7 and a quarter seconds to burn is required.

The best height for detonation of the bomb depends on the size of the bomb, and how hard the wind is blowing. With a bomb containing about 1 gallon of nerve gas, and winds between 10-20 mph, the best height is from 100 to 200 feet. With a larger bomb, or more calm conditions, a higher height is called for.

The best spacing for the bombs also depends upon their size and weather conditions, but for the standard conditions mentioned, a spacing of ½ mile to ¾ mile will give a solid coverage downwind.

Synthesis of Nerve Gas

The synthesis of nerve gas is not a project to be undertaken lightly. It is one of the most dangerous projects which can be done in the laboratory. The danger comes from the fact that a little bit on

the skin translates into a miserable death minutes later. Beginners at organic synthesis are notorious for spilling the things they are cooking onto themselves, so this is a job for a seasoned veteran. For this reason, a certain amount of chemical expertise will be assumed in the following section. It is meant to be fully understood by anyone who has made it through a couple semesters of college-level organic chemistry.

The care required in the manufacture of nerve gases can best be illustrated with a few stories from the World War II nerve-gas factories of Nazi Germany. These factories were busily churning out Tabun for most of the duration of the war. As to why they did not concentrate their effort on the much more potent Sarin, I can only venture two guesses. Either they found factory-scale production of Tabun much easier, or they were hung up on Tabun because cyanide is used in its production. Everybody knows how big the Nazis were on cyanide. It was certainly not a matter of not being familiar with Sarin, since their big man, Gerhard Schrader, led the team that invented them both.

The standard dress for the workers in the nerve-gas factories was a rubber suit made of two layers of rubber with a layer of cloth in between, and a respirator. Even with the protective clothing, most of the workers suffered from varying degrees of poisoning.

Accidents, of course, happened with the most deadly consequences. On one occasion, some pipefitters were working on a section of pipe when the pipe drained out Tabun on them. They all died within minutes. Another time, seven workers were zapped in the face with a gushing stream of Tabun. In spite of immediate and intensive medical effort, five of them died.

In a laboratory, accidents can be avoided much more easily than in a factory setting. Even so, protective clothing is definitely called for. A scuba wet suit with good rubber gloves (not surgical gloves because they're too thin) and face shield would be ideal. A bath nearby filled with a concentrated solution of bicarbonate of soda is another necessity. This is for washing off any spills that may hit the skin. A good hood for exhausting fumes to the outside has to rank

high on the list of priorities also. Add to this some atropine sulfate for emergency first aid. The antidotal dose is 4 milligrams by injection every 10 minutes until signs of atropine poisoning appear 2-PAM is also helpful.

Formerly, there were no laws prohibiting anyone in the United States from manufacturing nerve gas. Shortly after publication of the first edition of this book, however, Herb Kohl, one of my state's senators, introduced a bill to the Senate which made illegal production of nerve gas and other substances, such as ricin, which could be used as weapons of mass destruction. The penalty for doing so is now life in prison. Apparently these narrow-minded people consider the manufacture of nerve gas to be an antisocial act. As a result, it is best to not advertise the nature of the product too widely.

The equipment needed for production of nerve gas is basically the same as that needed to produce methamphetamine (see *Secrets of Methamphetamine Manufacture* by Uncle Fester) or any other of a wide range of organic chemicals. The standard distilling kit with a variety of sizes of round-bottom flasks is a must, as is a magnetic stirrer-hotplate and a good source of vacuum such as a properly working aspirator.

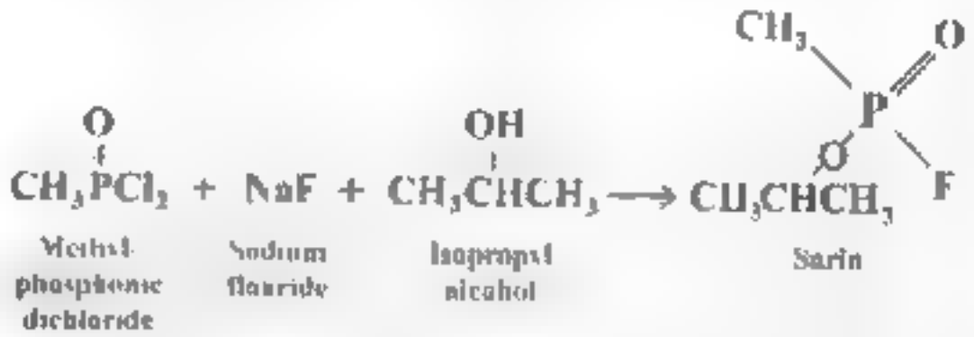
This section will concentrate on the manufacture of Sarin, with only passing reference made to the manufacture of the other nerve gases. There are several practical reasons for this. Sarin is considerably more potent than Tabun, yet not appreciably more difficult to make. Tabun uses in its manufacture the chemical whistleblower's delight, cyanide, so it is best to stay away from it. Soman is a little more potent than Sarin, but good, reliable directions for its preparation just have not been published, to the best of my knowledge. I will later make some speculations on its manufacture that I feel so sure of that I would bet my bottom dollar they are correct. Even so, it is best to stick to the sure thing. I'm sure you will agree with my logic.

Very good directions for the synthesis of Tabun can be found in an article by Holmstedt in *Acta Physiol. Scandinavia*, page 25 of

Supplement 90. Also of interest in the same article is a compound that is a few times more potent that Tabun called dimethylamido-isopropoxy-phosphoryl cyanide. Directions for its manufacture are found on page 27 of same article.

The synthesis of another also-ran nerve gas, called DPF, is described in detail by its inventor in a very fine book titled *Some Aspects of the Chemistry and Toxic Action of Compounds of Phosphorous and Fluorine* by B C. Saunders. More details of its preparation are covered in an article in the *Journal of the Chemistry Society of London*, page 695 to 699 (1948) by Saunders and Stacey. DPF is considerably weaker than Tabun, but the book is good reading as it covers how industrial-scale production problems were overcome.

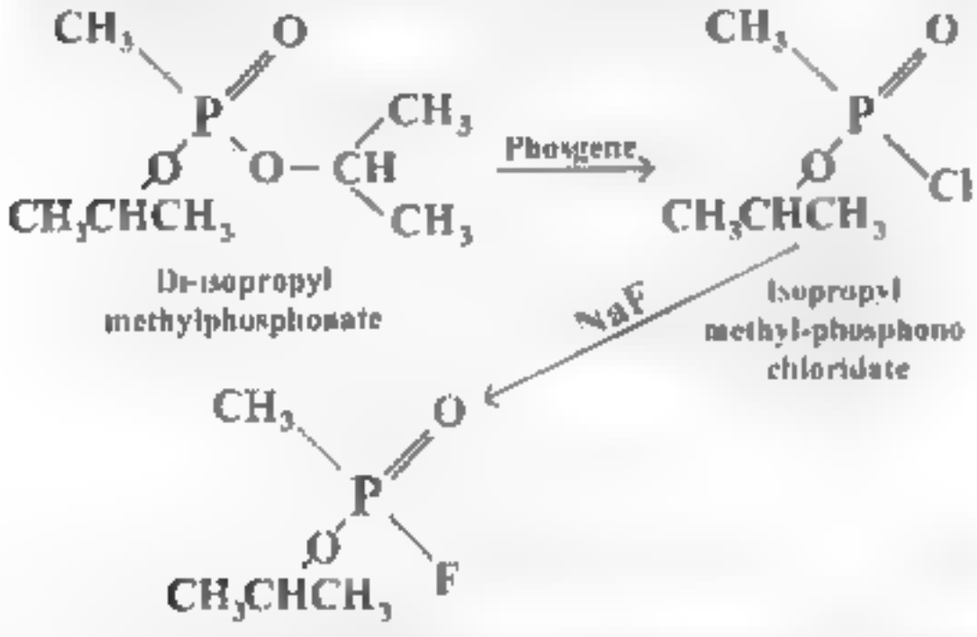
Sarin was made in Germany during World War II by the following process:



This method was workable, although the yields were low and the product was always contaminated with byproducts. Purity is next to Godliness even in nerve gas! This process could still be made to work today, since the starting material, methylphosphonic dichloride, is commercially available at the moment. It has to be a very suspicion-arousing material if anybody in the chemical industry has something on their minds other than dope. The price of a kilo is about $60. Directions for making it can be found in the *Journal of the Chemistry Society of London* page 3437 (1952).

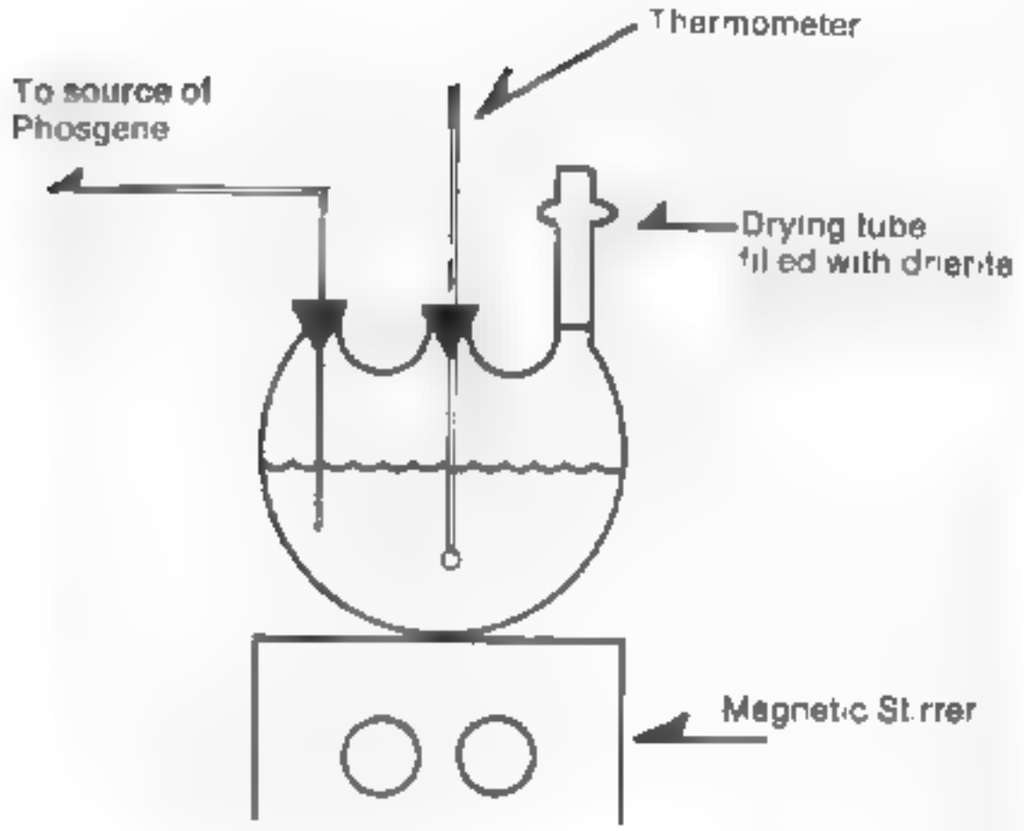
This process is described in detail by Schrader himself in *British Intelligence Objectives SubCommittee*, Volume 714, page 41 (1947).

A much better, although more tedious, process was reported in 1960 by a couple of Britishers named Ford-Moore and Bryant. The method they devised was not a simple "one-pot" process like the one that Schrader came up with. It is multi-step, with purification of each intermediate required. Their method is outlined below:



To the best of my knowledge, neither di-isopropyl methylphosphonate nor isopropyl methylphosphonochloridate are available commercially. This means that the process must start with cooking up a good supply of di-isopropyl methylphosphonate. Luckily, excellent directions for cooking it up can be found in *Organic Syntheses*, Volume 31, pages 33 to 35. The authors are Ford-Moore and Perry. It will not be reprised here because *Organic Syntheses* can be found in any good library, and the cooking directions in it are always so clear and reliable that one can place his utmost trust in them.

Now for the production of nerve gas. The glassware is set up as shown in the drawing on the next page.



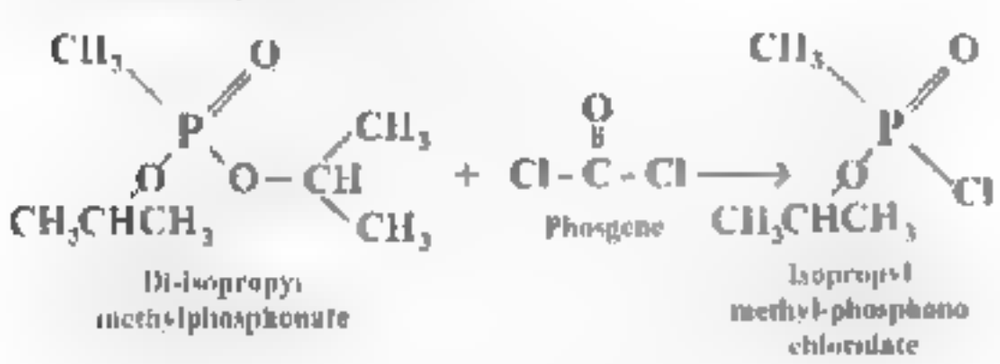
A 3-necked 1000 ml round-bottom flask is set up in the hood on a magnetic stirrer. Into the flask is placed 300 grams (304 ml) of di-isopropyl methylphosphonate. The flask should be baked in an oven for a few hours before use to drive out traces of moisture from the glass. This is very important for all glassware used in all phases of nerve-gas production because water reacts more easily with these chemicals than the desired reaction.

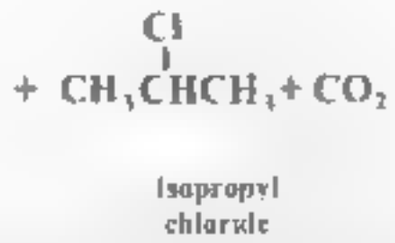
A drying tube is attached to one of the three necks. It is filled with drierite. NaOH should not be used because phosgene reacts with it. A magnetic stirring bar is put into the flask, and a thermometer is put down the central neck into the chemical in the flask to monitor its temperature. It is best held in place with an all-glass thermometer holder, but a cork may be used in a pinch. The use of rubber in any part of this assembly is to be avoided because phosgene attacks it, and will cause its remains to flow into the product. Through the third neck is put a section of glass tubing

connected to a cylinder of phosgene. The end of the tubing should extend well below the surface of the liquid in the flask. It may be held in place with a cork.

To begin the reaction, the magnetic stirring is begun, and a slow stream of phosgene is bubbled through the liquid. The thermometer is watched to make sure the temperature stays in the range of 20 to 30 degrees Centigrade. If the temperature creeps too high, it may be controlled by slowing down the bubbling and setting the flask in a bit of cool water. It is wise to check the efficiency of the hood while the reaction is going by puffing a cigarette outside the hood. It should suck the smoke in quickly. Smoke in the hood should never escape. Phosgene is a vicious poison; see the section on phosgene earlier in this book.

The following reaction takes place:





The bubbling of phosgene into the liquid is continued for 10 hours with stirring, then it is left to sit for some more hours, preferably overnight, or whenever the chemist is taking his sleep break.

When break time is over, a vacuum adapter is attached to one of the three necks, and the other two are stoppered. A vacuum is attached to the vacuum adapter, and a vacuum is slowly built up in the flask. An aspirator is best for producing the vacuum because it will flush the fumes down the drain. Stirring should be continued during the vacuum treatment. The vacuum will remove unreacted phosgene and the isopropyl chloride produced as a byproduct. Most of this unwanted material will be gone in about 10 minutes.

The vacuum line is then removed, and the reaction mixture is put in a 500 ml round-bottom flask, and the glassware set up for vacuum fractional distillation. A 10-inch-long Vigreux column works very well for vacuum distillations. An excellent substitute is to fill a claisen adapter about ¾ full with dime-sized pieces of broken glass. I have often used this for vacuum fractional distillations with fine results.

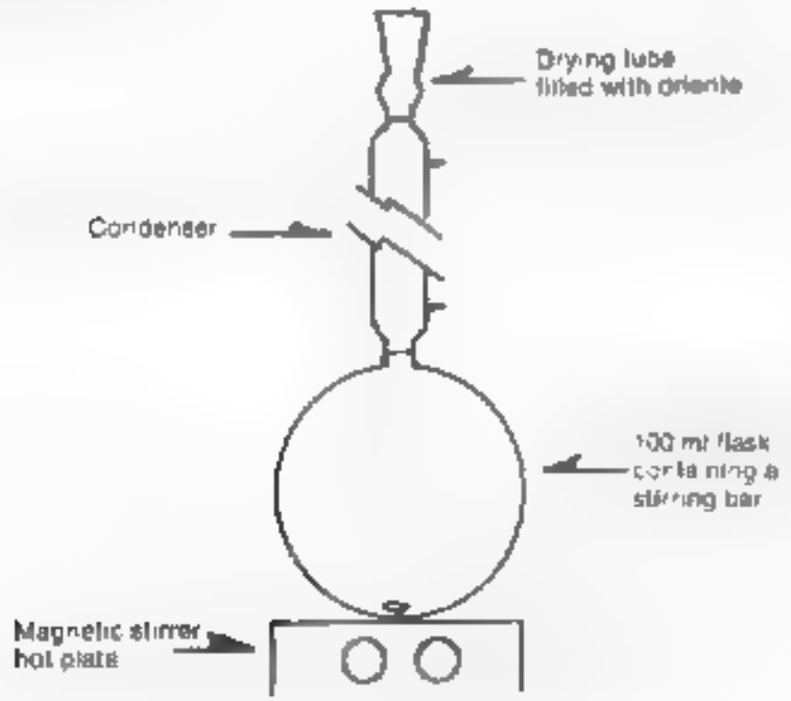
Distillation of the product is then commenced. I recommend a properly working aspirator as the source of vacuum. With a good, cold source of water to power it, a vacuum of from 10 to 20 mmHg should be attained. The small amount of forerun may be collected with a 50 ml round-bottom flask, then the product collected in a 250 ml round-bottom flask. The boiling point of the product at 2 mmHg is 38 to 40° C. And from 10 to 20 mmHg, its boiling point should be in the neighborhood of 55 to 60° C. The yield of clear liquid is nearly 250 ml.

A relevant cooking up and commentary are called for at this point. In the aftermath of the 1994 and 1995 Japanese nerve gas attacks, I spent in excess of twelve hours being interviewed by Japanese TV networks. During this time, I was able to pump them for information not available in this country. Analysis of the nerve-gas residue showed that a large fraction of their product was di-isopropyl methylphosphonate. This demonstrates, first of all, that the amount of phosgene they used in this reaction was shamefully inadequate. It further indicates that this fruit loop cult, made up of the "cream of Japanese society," didn't bother to distill the product

of the above reaction. If they had, they would have noticed that their reaction was incomplete at this step.

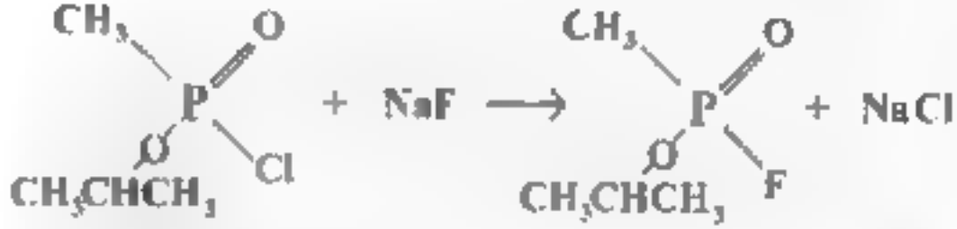
With the starting material made, Sarin can now be produced. Heavy-duty protective measures must now be taken by everyone involved in the synthesis of the final product. The starting material just produced, isopropyl methylphosphonochloridate, is not all that poisonous. It is only about as ponsonous as strychnine. The product of the next reaction, Sarin, is not so harmless. All work must be done in the hood. Rubber protective clothing must be worn. A 5-gallon plastic pail filled with water to which a few cans of lye have been added must be handy. This pail is for putting glassware into after they have been used. The strong lye solution will destroy the Sarin once it has been in contact with it for a few hours. A 6-hour soaking will make the glassware safe for further cleaning.

Now for how the Sarin is produced. The glassware is set up as shown below:



The glassware must be very dry to get the best results. Baking it in an oven for a few hours dries out glassware nicely Into the 1000 ml round-bottom flask is put 250 grams of the isopropyl methylphosphonochloridate made according to the directions above, and 250 ml of methylene chloride and 110 grams of sodium fluoride (NaF). All ingredients should be of a high grade and free of moisture. The stirring is begun, and heat applied to the flask until the mixture boils. Since methylene chloride boils at about 40° C, the water flowing through the condenser will have to be very cold to condense it. The boil is maintained at reflex for 4 hours.

The following reaction occurs:



Isopropyl methyl-phosphonic chloridate | Sodium flouride | Sarin | Table salt

After refluxing for 4 hours, the mixture is allowed to cool, and the stirring stopped. A Büchner funnel is then assembled to a filtering flask, and the flask is connected to the vacuum line. The filter paper is securely placed into the funnel, and wetted with some methylene chloride to hold it in place. The vacuum is turned on, and the reaction mixture is filtered. The salt which was made in the reaction will filter out. It should be rinsed with some methylene chloride to get the last of the product, Sarin, off of it.

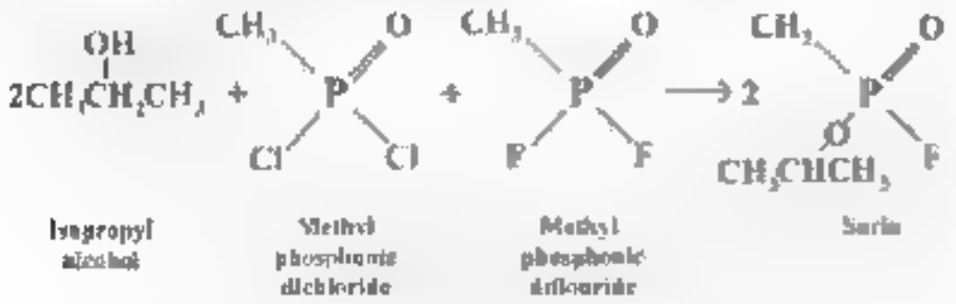
I must warn here that this filtering step, and the transfer to the distilling flask coming up, are the two most likely times for a spill accident to occur. Vigilance is the price of life!

The filtered product is transferred to a 1000 ml round-bottom flask, and the glassware set up for vacuum distillation. A few boiling chips (some pieces of pumice foot stone make a good substitute) should be in the flask. A Vigreux column or similar fractionating device should be attached.

A vacuum is applied, and the methylene chloride is removed under a vacuum. When most of it is gone, heat may be applied to the flask and the product collected in a 250 ml flask. Sarin boils at 56° C at 16 mmHg and 46° C at 8 mmHg. The yield is a little over 200 ml of Sarin.

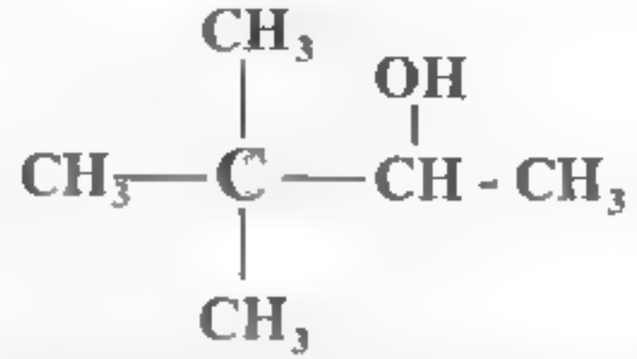
All glassware goes into the lye soak after use, and the rubber clothing into a somewhat weaker lye bath. Work surfaces should also be drenched with the lye solution. The end of the vacuum line must also be soaked. The container that the Sarin is ultimately put into should be very securely stopped when full, and then sprayed down on the outside with a lye solution. A plastic plant mister works well for this. After rinsing off the solution after a few hours, the container should be coated with a layer of wax. It may now be considered safe, so long as it is not dropped.

Now for my speculations on the production of Soman. See the *Journal of the Chemistry Society of London (1960)*, pages 1553 to 1555 by Ford-Moore and Bryant. In addition to the above method of Sarin production, they list another one which would be tailor-made for the production of Soman. Their method is:



Isopropyl alcohol | Methyl phosphonic dichloride | Methyl phosphonic difluoride | Sarin

If, in this process, pinacolyl alcohol was substituted for isopropyl alcohol, the product would be Soman. Pinacolyl alcohol has the structure:



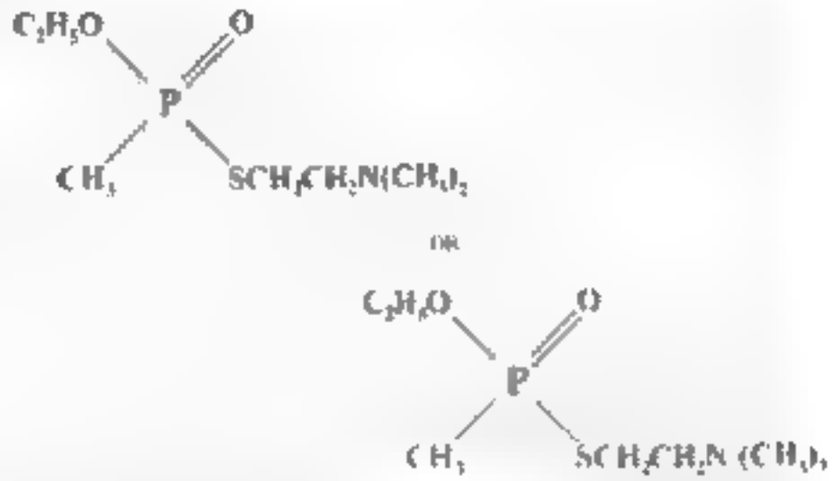
It has the IUPAC name 3,3,-dimethyl-2-butanol, and sells for $40 per 100 grams. It may also be obtained by reduction of pinacolone. Most of us are familiar with this substance from doing the pinacol rearrangement in organic lab.

Another possible route to Soman would be to substitute pinacolyl alcohol for isopropyl alcohol in Schrader's original method for making Sarin. Of course, it goes without saying that since pinacolyl alcohol has a molecular weight of 102.2 versus isopropyl alcohol's MW of 60.1, it would be necessary to use at least 1.7 times as much pinacolyl alcohol as would be needed with isopropyl alcohol. Even more may be needed, if, as I suspect, pinacolyl alcohol is much less reactive than isopropyl alcohol.

The V-Gases

Sarin and Soman were not the end of the line for nerve-gas development. Better killing through chemistry brought forth a new series of improved nerve gases in the late '50s and early '60s. This series of improved nerve gases have chemical structures very similar to Sarin (and are made almost the same way), but possess some relatively minor modifications that increase their potency considerably. The particular V-gas which will be concentrated upon

here is the most potent of all the V-gases. Its chemical name is methylethoxyphosphoryl thiocholine and has the following structure:



This compound is 10 times more potent than Sarin, and yet is not appreciably more difficult to make than Sarin. The advantages of the more potent product are obvious for the large-scale attack. The one-gallon size blast-dispersal bomb described earlier in this chapter, if filled with this V-gas, would then become the equivalent of a 10-gallon bomb. The lethal zone downwind of the drop sites would be considerably greater with the V-gas. The same size payload on the bomber plane would pack a much greater punch.

The two chemical structures shown above are both correct. The one on the left is the thiocholine nerve gas as it is produced in the main synthesis reaction. The one on the right is the quaternary ammonium compound derivative produced from it by reacting it with methyl iodide. The quaternary ammonium compound has the advantages of being more potent (it is believed that the positive charge on the nitrogen atom makes the poison more efficient at linking up with and deactivating the cholinesterase enzyme), and also more suitable for use as an assassination poison.

The quaternary derivative is more suited for assassination because the quaternary ammonium compounds are notoriously hard to detect in the body of the victim at autopsy. A V-gas

assassination is likely to remain a mystery, so long as it is done stealthily. Of course, if an anti-cholinesterase test is done on the blood of the mark, it will show that *something* has knocked out this enzyme system, but finding out what will be a task for a magician. This test would have to be done while the body is still fresh, or the enzyme system will go kaput anyway as a natural result of being dead and rotting.

The unquaternized product as it comes out of the reaction has the advantage of being a liquid (the quaternized material is a solid) so it is more easily dispersed into a fine mist of droplets from a blast-dispersal bomb, or an aerosol dispenser. A larger explosive charge would be necessary to do the job of shattering a solid mass of V-gas into a mist.

The unquaternized product has the disadvantage of not keeping as well as the quaternized product. It tends to rearrange into less-toxic substances when left standing. This process can be slowed down by freezing, but not stopped completely. In any case, it is very slow and not a matter for concern.

The V-gas, in both its unquaternized, and especially its quaternized form, has one great disadvantage in comparison to Sarin or Soman. It is not possible to vaporize the V-gas by heating it. This means that one possible means of attack by nerve agents is not going to work with the V-gas.

The attack plan to which I am referring here would be an option when a large group of the enemy is meeting in an auditorium, arena, coliseum or similar large structure that is under cover. In this example, the enemy could be attacked here with Sarin simply by heating a reasonable amount (a couple of pounds) of Sarin above its boiling point (nearly 200° C) in a location where the ventilation system would rapidly spread the gas throughout the building. A pandemonious scene reminiscent of spraying a room filled with flies would be the most immediate result. About half the Sarin would be lost to charring from heating it in this manner without the protection of a vacuum, but that is a trade-off likely to

be accepted by the attackers because of the simplicity of this means of attack.

A variation of this attack plan was used by that whacked-out Japanese cult in their first nerve-gas attack in the city of Matsumoto about six months prior to their subway escapade in Tokyo. The controlled US media blacked out news of this attack for fear of inspiring copycats, so let me fill you in on the details. In this trial run for the big show, some unseen operatives pulled into a parking lot a little upwind from an apartment complex. There they rapidly heated maybe a pint of their Sarin product, and let the fumes drift downwind into the apartments, where they killed about a dozen people and wounded 200. An interesting sidelight here was the behavior of Sarin when rapidly heated to boiling without benefit of a vacuum to protect it. White clouds were seen drifting on the winds into the apartments, these clouds no doubt being oxides of phosphorus formed from the breakdown of the Sarin due to heat. Also, vegetation in the vicinity was scorched, due to action of the phosphorus oxides, along with HCl and HF.

This reasonably effective, although hardly inconspicuous delivery method was abandoned in favor of a crackpot scheme for their subway attack. For this event, the aforementioned "cream of Japanese society" decided to dilute their Sarin with acetonitrile, put it in plastic bags hidden in luggage, and just poke the bag to puncture it and spill the solution onto the floor of the subway. To get the Sarin up into the air, they just relied upon passive evaporation, a very slow and ineffective process with a high-boiling-point substance such as Sarin. Even with the added surface area imparted by the dilution with acetonitrile, Sarin's vapor pressure is just too low at room temperature to cause much to evaporate into the air.

To attack a target such as the subway, these "geniuses of the Tokyo tunnels" would have been better advised to use an aerosol dispenser. Such a device could have simply enough been constructed and used by these aforementioned "geniuses." To begin

to consider this alternative, one must first look at and understand the common aerosol can found in all our homes.

Pictured above is a typical aerosol spray can. The can is filled with a solution of propellant and product. The purpose of the propellant is to create a good level of pressure inside the can to force the mixture up the stem and out the spray nozzle. There the propellant rapidly evaporates away, leaving a fine mist of the product drifting in the air. For nerve gas, one can use a very similar design. There are no real limits upon the possible size of the aerosol container. The preferred material for construction of this device is stainless steel, as fairly thin layers of steel can be eaten away by nerve gas, which could lead to the rupture of the vessel. The preferred propellant for nerve gas is Freon 12.

To construct such a device, one should start with the stem. It should reach nearly to the bottom of the container, and be welded flawlessly unto a lid or filling portal for the container. Where the stem leaves the container, there should be two high-quality valves, such as from Swage-Lock, in series to hold back the product in the

can once it is filled. The stem should end in a spray nozzle such as those found at the hardware store for garden sprayers.

Next, we should consider filling the container. When filled, one would want about ⅔ of its volume taken up with propellant-product solution, and one would also want the solution to be about 10% nerve gas and 90% propellant Freon 12. So, for a one-gallon container, add 250 ml of nerve gas. Cool down the container bottom with dry ice-acetone, and similarly chill a container of Freon 12. The boiling point of Freon 12 is -30° C, so it must be well below that temperature. Then, when it is sufficiently cold, drain the liquid Freon from its tanks into the aerosol container until it is about ⅔ full. Then weld the top or filling portal with the spray stem onto the container. This completes construction of a nerve-gas aerosol can. It can be checked for leakage once it warms back to room temperature by using caged birds, etc. Finally, an automatic system which opens the valves on the stem is easily constructed. It would be most convenient to rig this automatic opening system with a timer, although radio control is also possible.

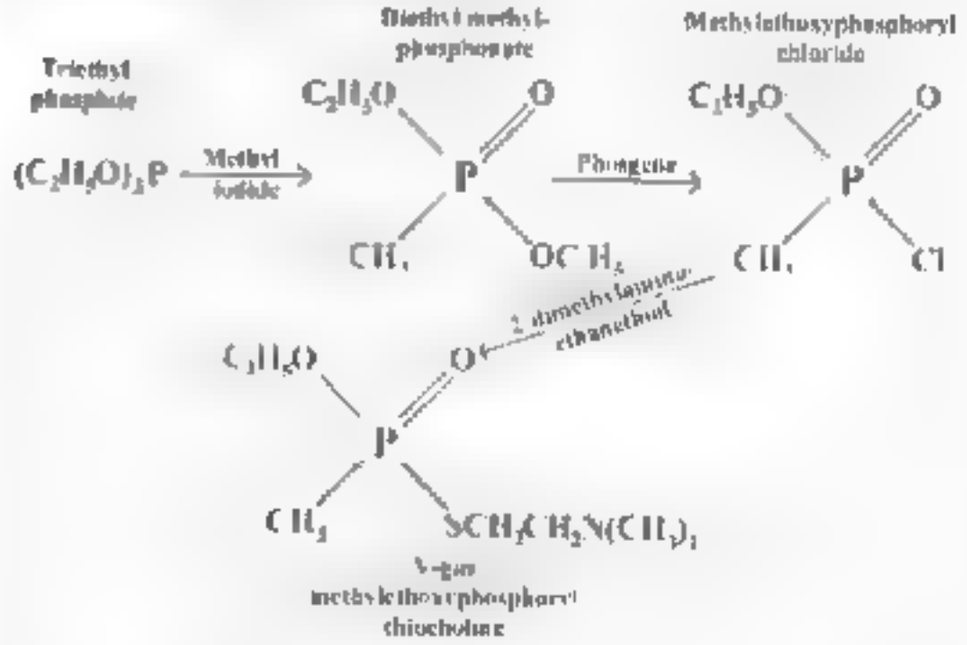
The synthesis of V-gas is very similar to the synthesis of Sarin. No special equipment is needed to produce it, just standard chemical glassware with ground-glass joints. One change from the synthesis of Sarin is that a good quality vacuum pump is required to produce a vacuum for the distillation of the final product. It has such a high boiling point that a vacuum less than 5 mmHg will have to be applied to distill it without burning the product.

The starting material for the production of V-gas is triethyl phosphite (for Sarin it was triisopropyl phosphite). This substance costs about $15 per pound. An attacker may be wise to consider making this starting material, because the federal government tries to keep an eye on sales of materials which can be used for nerve gas manufacture. How well they do this, with all the attention they lavish on dope, is a matter for speculation. Whether triethyl phosphite is a remote enough precursor to evade their watchful eye, one can only guess. In any case, the main manufacturers would be bad places for an attacker to purchase this material because they

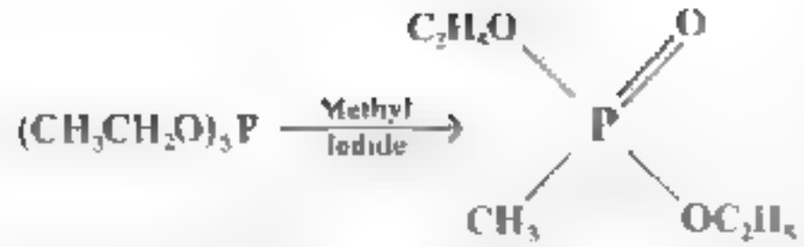
are all notorious government snitches to protect their federal contracts. The smaller local distributors may be less enamored of filling out forms for Uncle Sam. Very good directions for making triethyl phosphite can be found in *Organic Syntheses*, Volume 31, or Collective Volume 4, page 955.

As you can see by comparing this process to the Sarin production process, they are almost identical. Instead of triisopropyl phosphite as the starting material, we have triethyl phosphite. From there the process is the same until the final step where sodium fluoride is replaced with 2-dimethylaminoethanethiol.

Starting with triethyl phosphite:

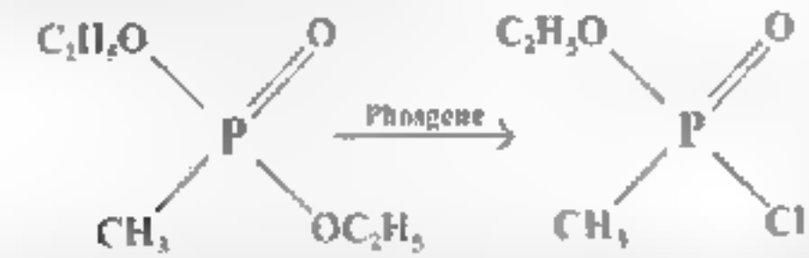


Directions for the first reaction can be found in *Organic Syntheses*, Volume 31 or Collective Volume 4, page 325. The title of the synthesis is Di-isopropyl methylphosphonate, which was the material used for the Sarin synthesis. At the end of the article, at the bottom of Note 2, is the modified direction for making diethyl methylphosphonate:



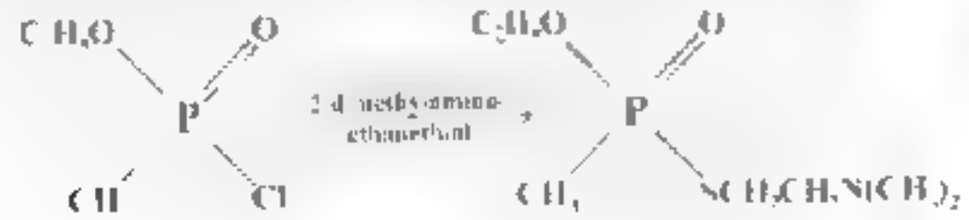
Equal molar amounts of methyl iodide and triethyl phosphite are reacted. For a convenient and productive 2-mole-size batch, that means that 332 grams of triethyl phosphite (348 ml) is added to 284 grams (126 ml) of methyl iodide. They are refluxed together for three hours, then the purification procedure given in the main text of the article is followed. The authors note that a small amount of a byproduct, diethyl ethylphosphonate is made, and that it is very hard to separate it out by distillation. It is my opinion that this small amount of byproduct can be lived with, and need not concern an attacker worried about the success of the next reactions.

The next reaction is:



- This reaction is done exactly the same way as in the Sarin synthesis. The phosgene (a.k.a. carbonyl chloride) is bubbled through the material for 10 hours, and the product is recovered by distilling.

With the preliminaries out of the way, the nerve-gas-crazed attackers are ready for the main production reaction:



Naturally, the same safety precautions for the Sarin synthesis are needed for this reaction. Since an aspirator cannot produce the vacuum needed for the distillation of the product, it is doubly important that the fume hood used for this reaction have a good, steady draft of air moving through it. The vacuum pump must be in it, so that its exhausted vapors do not fill the room.

The interesting substance used instead of sodium fluoride, 2-dimethylamino ethanethiol, is another chemical that a careful attack group may consider making themselves rather than purchasing ready-made. I feel that this decision is wrong because the substance has legitimate use as a metal-complexing agent, and so with subterfuge could probably be obtained without arousing suspicion.

Weighing on the other side of the argument is the very high cost of this material. Aldrich is selling it for over $50 per hundred grams, as the hydrochloride, with a purity of 90%. So to use it, it would have to be free-based and purified. The substance it is made from, N, N-dimethylethanolamine, is sold by the same folks at 99% purity, for about $15 per kilo. This substance finds use in biology and medicine as an inhibitor of ethanolamine phosphokinase. This alcohol can be turned into the thiol by first reacting it with $SOCl_2$ (see *Journal of the American Chemical Society*, Volume 66, page 1921 (1944) by Breslow), and then reacting that with alkaline hydrosulfite solution under inert atmosphere (see *Journal of the American Chemical Society*, Volume 67, page 1845 (1945) by Gilman).

In any case, the main production reaction for V-gas is surprisingly easy. It is done as follows:

Into a clean, dry 2000 ml round-bottom flask, the following ingredients are added, quickly, one after the other with swirling to mix them: a few boiling chips, 800 ml anhydrous ethyl ether, 284 grams of the methyl ethoxyphosphoryl chloride, 212 grams of dimethylaminoethanethiol, and 212 grams (292 ml, cost about $15 per kilo) of triethylamine. It is crucial here that the glassware be very dry, and that the ingredients, especially the chloride, be protected from moisture, because the presence of water really

lowers the yield in this reaction. I recommend baking the glassware in an oven, and allowing it to dry in a dessicator

When the ingredients have been added and mixed, a good, efficient condenser topped with a drying tube is attached to the flask, and a flow of good, cold water is put through the condenser. The contents of the flask are heated to boiling with a hot-water bath, and a reflux is maintained for one hour.

The byproduct of this reaction, hydrogen chloride, is absorbed by the triethylamine as it is produced, forming triethylamine hydrochloride crystals. This acid absorption is the reason for having the triethylamine in the reaction mixture. Without it, the hydrogen chloride evolved would interfere with the reaction.

At the end of the one-hour heating period, the mixture is cooled, and the crystals of triethylamine hydrochloride are filtered out in a Buchner funnel. The crystals may be rinsed with ether to get the last of the product off them.

The filtered reaction mixture is then returned to a 2000 ml round-bottom flask, a few boiling chips added, the glassware set up for simple distillation, and the ether removed by distilling it off under a gentle vacuum. An aspirator is perfect for this job, since it will flush the ether fumes down the drain.

When most of the ether is gone, the mixture is poured into a 1000 ml round-bottom flask with a few boiling chips. The remnants in the 2000 ml flask can be rinsed out with some ether and poured into the 1000 ml flask. Once again this flask is set up for simple distillation, and full aspirator vacuum is applied to it. The last of the ether and triethylamine (bp 88° C) will be gone shortly.

Now the vacuum from the good-quality vacuum pump is applied to the distillation. A vacuum of less than 1 mmHg is to be preferred here to keep the distillation temperatures reasonable and to avoid burning the product. A McLeod gauge should be used to keep track of what kind of vacuum the pump is pulling (cost about $200).

After a small forerun is collected in a 250 ml flask, a 500 ml flask is attached, and the main bulk of the product is collected at a boiling point of 80° C at a vacuum of .06 mmHg. This boiling

point rapidly increases with increasing pressure. The yield is 260 to 275 ml of product. A fair amount of tar remains in the distilling flask.

This product may be poured into a large glass bottle for storage, as with Sarin. The 40 oz. beer bottle is ideal for bomb design, and once it is coated with wax, it is fairly safe, if not dropped.

If the aim of the attacker is a single assassination, quaternization is called for. To do this, a couple drops of V-gas are put into a test tube ¼ filled with ether. Twice the number of drops of methyl iodide as V-gas is then added, along with a boiling chip. The mixture is heated to boiling for a few hours, then allowed to slowly cool down. The ether and unreacted methyl iodide are removed under a vacuum, the crystals washed with a little more ether, and the resulting product is ready for use.

Chapter Five
TIME DELAY FIRE

Fire has been one of man's favorite weapons of destruction for the thousands of years it has been at his disposal. Neither the passage of time, nor the advance of technology has done anything to take the glimmer off this ancient bringer of death. It is still the silent friend of the angel of death.

Those who would use fire as an instrument of destruction, or a means of assassination, have two large stumbling blocks in their way. To achieve success both must be surmounted.

The first obstacle to be overcome is that it may prove helpful to the attacker if he can prove he was "somewhere else" at the time the fire broke out. Even occupying forces battling guerrilla fighters can often be swayed by the powerful logic of a good "alibi." Its effect in more normal situations can be overwhelming.

The second obstacle is not so easily passed. That is to make the tragic event appear to be of natural origin or an accident. Here the matter becomes touchy because the "torch" runs up against the accumulated wisdom of fire marshals and inspectors. Backing them up is their ever-present ally, the crime lab with its gas chromatograph.

A well-trained fire marshal has the uncanny ability to tell where in a building a fire started, just by "reading" the pattern of the char markings on the wreckage. If the fire is traced to an area of the building where there should be no source of fire to start the blaze, alarm bells go off.

An even more amazing fact is that the charred remains from a fire can retain traces of any "accelerant" used to start it. Lab equipment has progressed to the point that if gasoline was used as the accelerant, it is easy to tell whether it was regular or unleaded. It may even be possible to tell the brand.

Entering this scene is a very widely known chemical with a very little known property. Hydrogen peroxide (H_2O_2) is an everyday item, often seen in medicine cabinets or at the drug store. It is usually sold for home use as a 3% solution of hydrogen peroxide in water. Other concentrations of hydrogen peroxide, ranging from 20 to 50% hydrogen peroxide are standard items of commerce. They find use in industry as bleaching agents, and in the laboratory as oxidants and as means to make chemicals.

These stronger solutions of hydrogen peroxide have the interesting property of setting fire to things. They don't do it right away, and they won't set everything on fire.

Flammable items like wood, cloth, furniture, carpeting, paper, etc., are all easy pickings for hydrogen peroxide. Anything that will normally burn, and that hydrogen peroxide can soak into, it will set on fire.

The amount of time delay between applying hydrogen peroxide and the resulting fire depends on a lot of factors. This is because the water in the hydrogen peroxide solution must evaporate away, allowing the hydrogen peroxide content of the solution to get higher and higher until a critical point is reached and the item bursts into flame. So the warmer and drier it is, the faster the water will evaporate and the faster it will burst into flame. A typical time delay for a few ounces of 30% hydrogen peroxide solution doused onto cloth is about an hour at a temperature of 70° F and a humidity of 40%.

The more concentrated solutions of hydrogen peroxide look pretty much like the 3% brand seen in the drug store, which is to say they look like water. They also have no smell, until they begin to smolder just before ignition.

How this ties into stumbling block number one is pretty obvious. What about number two? Hydrogen peroxide is a pretty unstable substance at high temperatures. During a fire, it will break down to water and oxygen. Most detectors on gas chromatographs will not even know it is there, if it should survive the fire. Finally, if it should be detected, would the people in the lab know what it means?

Chapter Six
POISONS FROM NATURE

Digitalis

Most of us are familiar with digitalis, the life saving heart drug taken by millions of people around the world. Many of us have also seen its use in movies and TV as a means of "untraceable assassination" used by hit men, etc., to cause mysterious heart attacks.

Like most of what one receives from the popular media, this is pure bunk. While it is true that an overdose of digitalis will cause what at first glance appears to be a heart attack, it is by no means untraceable. In truth, digitalis is rather easily detected in the human body. It is far from mysterious, or even clever. A wide variety of methods have been devised to detect digitalis and all of the so-called "cardiac glycosides."

There is, however, one exception to this general rule which groups digitalis with the "bad poisons." That is if the mark is already taking digitalis by prescription. It certainly is ironic that taking digitalis makes one vulnerable to poisoning, both accidental and otherwise from it. A good doctor will closely monitor the blood level of digitalis in the bodies of his patients, precisely because sudden shifts in body chemistry can cause blood levels of digitalis to rise to the lethal point without changing the dose involved.

The lethal level of digitalis is about 3 or 4 times higher than the amount used to improve heart function, so there is not a large safety zone in which the blood levels of digitalis can move.

For this reason, it is next to impossible for a coroner to say, just on the basis of blood levels, whether the dear departed was the victim of malice or unfortunate swings in metabolism. (See *Advances in Forensic and Clinical Toxicology*, by A.S. Curry, page 158.)

This is not to say that other factors might not tip him off. For example, if the remains of a couple Foxglove plants (the source of digitalis) are found in the stomach of the mark, doubt may arise whether or not he knowingly or willingly ate them.

Foxglove, a fairly common ornamental plant, is the natural source of digitalis. It is found in the leaves, seeds, and flowers of the plant. The digitalis can be extracted from the plant by blenderizing the plant parts containing the digitalis until they are powdered, and then soaking the powder in vodka (100 proof will do, but 190 proof is better), filtering the resulting brew, and allowing the alcohol to evaporate. The residue contains digitalis. A much better job can be done using an Iso-II hash oil extractor. I always used to see them advertised in *High Times*, but I haven't seen an ad lately. Perhaps one could be found at a garage sale. Like the gas generator, the Iso-II is another piece of equipment that no home is complete without.

The extract so obtained will contain a mixture of digitalis glycosides, mainly digitoxin, gitoxin, and digoxin. Modern medicines do not usually contain a mixture of ingredients. For this reason, prescription medicine matching that of the mark is employed by successful poisoners.

For more information see: "Fluorometric Determination of Myocardial Digoxin at Autopsy with Determination of Digitalis Leaf, Digitoxin, and Gitoxin" by R.W. Jelliffe in the *American Journal of Clinical Pathology*, Volume 51, page 347 (1969).

Rapeseed Oil Poisoning

In 1981, a mystery disease swept through Spain. The unfortunate victims of this bewildering malady seemed to age decades in a matter of days. Their immune systems turned against their own bodies and chewed what was left of them to bits. They became steadily shrinking human skeletons, except for their rapidly growing tumors. The nervous system was also a target, resulting in loss of control of muscles for the lucky. Those not so fortunate were cast into the depths of insanity. In a matter of months, 12,000 people were stricken. Most are now dead.

As one would expect, the best minds and equipment were marshaled to find the cause of this mysterious illness, but try as they may, nothing could be found. Blood was analyzed and urine was scrutinized, and every available orifice was probed, to no avail. Every test came up negative. The best minds were stumped.

Luckily for those not yet stricken, another group was on the case. They were like medical detectives, questioning each victim who could still talk, and their relatives. They inquired into every small detail of their lives, looking for the common thread which bound them together in misery.

After several months, they hit the jackpot. It turned out that all of the victims had been using a "bargain brand" of "olive oil" sold by some shady dealers. Little did the victims know that this "bargain brand" was not olive oil at all, but instead was industrial grade rapeseed oil.

The industrial grade rapeseed oil was definitely not meant for consumption by any living thing. It was meant for use as a drying oil in paints instead, so it had been processed in a manner similar to boiled linseed oil to increase its polyunsaturated fat content. These polyunsaturates are notorious for their ability to generate free radicals. In a paint, these free radicals allow the paint to polymerize and "dry." Inside a living body, these free radicals react randomly with living tissue, aging it. After enough damage is done, the

immune system no longer recognizes the damaged tissue as belonging to the body, so it attacks it. In no time at all, the whole organism falls apart in a most mysterious manner. Keep that in mind next time some product boasts of its polyunsaturated fat content.

One would think that this industrial grade rapeseed oil would be pretty unpalatable. Apparently, that was not the case, as all those thousands of erstwhile bargain hunters eagerly chewed down the oil, and came back for more. From this, one can conclude that its smell, taste, and appearance were pleasing to the unfortunate victims of the "mystery disease." Logic would then dictate that it could be incorporated into the diet of almost anyone, so long as it were mixed with some of the genuine oil it is replacing. For instance, I'm sure that Wesson could be cut with this rapeseed oil 50-50, without arousing suspicion from those partaking in this unhealthy repast.

Rapeseed oil is a fairly common item in the paint industry. It is one of a large number of plant oils which have been used as "drying oils" in paint. Other members of this group include linseed oil and tung oil. A good paint supply center should be able to get some if they don't have any on hand.

To the best of my knowledge, the lethal dose of industrial grade rapeseed oil has not been determined. It is a pretty good guess that the lethal amount would be near half a cup. Lower amounts can be counted on to do heavy damage as well. Chemicals which form free radicals work in much the same way as radioactive materials. There is no safe level of exposure, merely a level at which the risk becomes acceptable.

The Jequirity Bean

The jequirity bean, also called the rosary pea or crab's eye, is a vine which grows in tropical areas, including Florida. This plant produces beautiful seeds which are popular with the local craftsmen

for fashioning necklaces, rosaries, and similar objects. Behind this innocuous facade, the jequirity bean hides a sinister secret. It contains a very deadly and difficult to detect poison.

The beautiful, yet deadly seeds of the jequirity bean are about the size of a pea, and are red and black in color. Their hard coats make them perfect for making jewelry, and in pea shooters, I suppose. Their beauty also makes it very unlikely that anyone would question the desire of a person to own a few pounds worth of these little gems.

Beneath the hard coat of the seeds, in the pulp of the seed, is a most fascinating poison called abrin. Abrin is an unusual plant poison, because it is a protein (technical term: phytalbumin), in contrast to the great majority of plant poisons, which are alkaloids. This is a very important point because all the alkaloids can now be easily detected and identified by use of a gas chromatograph mass spectrometer. Formerly excellent poisons such as nicotine or aconitine are now virtually useless because of the ease of detection of tiny quantities of poison possible with the GC-mass spec. Proteins, on the other hand can be very difficult to detect in a body. This is because of the needle in the haystack effect. With all the thousands of different proteins in a body, to pick out one that doesn't belong there is very difficult, especially if it is not there in large quantities. Another class of compounds that share this quality of difficult detection are quaternary ammonium compounds.

The jequirity bean has more to recommend it than just difficulty of detection. The timing of the symptoms are also very convenient for successful poisoning. The symptoms come on at least a day after eating the jequirity bean, and may take as long as 3 days to begin.

This is helpful because it makes it pretty difficult for the mark to associate his distress with its cause a few days earlier. There are two "good" extremes for time of action of a poison. Either quick knockdown with immediate death, or time delay effect, the longer the better.

The symptoms of poisoning by the jequirity bean are not very helpful in diagnosing the illness. The victim suffers from vomiting, diarrhea, and finally collapse and death. Symptoms like these are seen in so many illnesses and poisonings that they are no help.

The fatal dose of jequirity bean is about 10 of them for an adult. The wise poisoner will add a few to be sure of the results. In preparing the beans for serving, there are several points which must be observed for best results. Point number one is that the beans must be completely hulled. There are two reasons for this. The first reason is that the poison is locked beneath the hard shell of the pea, so the shell must be cracked for it to get out. The second reason for husking the peas is that intestinal contents, stomach contents and vomit and feces are usually closely examined for clues to the illness or death of the unfortunate victim. Finding the hulls of jequirity beans in any of the above mentioned materials would be a dead giveaway to the cause of the malady.

Serving the jequirity bean is sure to be an art form all to itself, but I know of no books here to recommend on the topic. I can offer two guidelines. Most importantly, care must be taken in heating the seed contents. Proteins are sensitive to cooking, and their properties are radically changed in the process. This process is called denaturation, and is irreversible. A prime example is what happens to the egg white (protein?) upon frying. It is a good bet that cooking destroys the poison in the jequirity bean. Point number two is a serving suggestion: mushed up and mixed with mashed potatoes.

Chapter Seven
CIA SHELLFISH TOXIN

We've all seen spy movies where one spy does in another with a small poke from a pin, or a droplet in a drink. The victim always falls over dead in a flash, never making a sound other than a grunt. The CIA shellfish toxin is the real life counterpart of the spy movie's poison.

The CIA shellfish toxin, known also as saxitoxin or gonyaulotoxin, is one of the most diabolically effective poisons for assassination known. It is effective within seconds when injected. When eaten the symptoms take longer to begin, but its lethal fury will not be denied, regardless of whatever medical assistance may be available.

This toxin is also exceedingly difficult to detect and analyze in the body of the victim. There are several reasons for this. First of all, saxitoxin is a very mysterious and elusive substance. It does not have many easily definable chemical characteristics. Scientists have been studying this stuff for years, and they still can't agree on just what its chemical structure is. Recent studies seem to have narrowed the scope of the argument to a few possibilities, but people keep on publishing new papers disputing the claims made just a few years before.

Saxitoxin does not seem to be suitable for analysis by the GC-mass spec (see Chapter Thirteen for more details), so it evades detection by the most powerful and commonly used analysis machine in forensic toxicology. All the published articles that I

have read used liquid chromatography to isolate and detect saxitoxin. This more troublesome method is often not used, and it must be coupled with a chemical test for identification. Saxitoxin's lack of characteristic chemical reactions makes detection by this method unlikely unless there is some other clue to help them along, such as undigested shellfish in the stomach.

Saxitoxin is such a powerful poison that tiny amounts are quite deadly. With such a tiny amount in the body of the victim, it gets diluted to the point where it is almost lost. This makes the task of finding it that much more difficult.

Exactly how poisonous this shellfish (or mussel) poison is depends upon how it is administered to the mark. For a 175 lb. man, 5.3 milligrams is required by mouth, .068 milligrams (68 micrograms) by intravenous injection, and .2 milligrams when injected intraperitoneally (into the stomach muscles like the old rabies vaccine). This large difference is caused by the fact that it is rather slowly absorbed into the body from the intestines, and rapidly excreted by the kidneys, so a much larger amount of poison is required when given by mouth than by other means.

The symptoms of saxitoxin poisoning when it is given orally are interesting to say the least. Within 30 minutes of eating it, a numbness or burning is felt in the mouth. This spreads over the rest of the head, and then to the finger tips and toes. The victim then becomes uncoordinated and appears to be drunk. Words are formed only with the greatest difficulty, and they are usually too slurred to be understood. Difficulty in breathing comes next, and if a fatal dose has been taken, death ensues within a few hours. If given by injection, the course of events is much faster, (within a few minutes) but the cause of death is still respiratory failure.

The source of shellfish toxin is clams and mussels growing in coastal areas. Along the shore of the Gulf of Mexico, a slightly different toxin is involved, but it is just as deadly. These clams and mussels become poisonous during the summer months because they filter out and feed upon poisonous plankton belonging to the genus Gonaulax and Gymnodinium. These plankton contain the saxitoxin,

and the clams and mussels concentrate it in their bodies after feeding upon it. It does the clams no harm, but can bring death to any warm-blooded animal eating the clam while it is still contaminated.

The exact amount of saxitoxin in a given clam or mussel is very hard to predict. It depends on how many of the poisonous plankton have been growing nearby. Blooms of these deadly plankton often color the water red or brown, and are responsible for the infamous "red tides." A study was done to follow the toxicity of the clams and mussels during the course of the summer, and they found that the poison content can increase and decrease by a factor of ten over a two week period. The peak of deadliness is usually reached during July. Clams such as the Alaskan Butter Clam remain poisonous for a period of almost a year after feeding upon the poisonous plankton, while mussels like *Myrtilus californianus* flush the poison out of their bodies rapidly and may be safe after a few weeks away from the deadly plankton.

As could be guessed from the above, it is pretty hard to say just how many mussels or clams will provide a lethal dose to the target. A few generalities here can make life much simpler. First of all, mussels are usually much more toxic than clams. *Myrtilus californianus*, which grows all over the California coast, is an especially good concentrator of the poison. An average summer will result in 4 or 5 mussels being the amount required to be eaten to cause symptoms, and over 12 being the lethal dose. After putting away that many shellfish, even Jethro Bodine would be full. That is why most cases of shellfish poisoning aren't fatal. A quirk of fate, however, simplifies things again. Over two thirds of the poison in clams is concentrated in the siphon. In mussels, over 90% of the poison is found in the digestive organs (the dark meat). This greatly cuts down on the amount of shellfish which must be consumed for fatal effect. All the attacker needs to do is some dissection with a sharp knife to be sure that only the most poisonous parts are used.

In many ways, feeding the shellfish directly to the mark is the best way of delivering this poison. It is very simple and requires no kind of skill. Cooking does not effect the poison, so it can be served in a variety of dishes. The poison dissolves well in the broth, so clam chowder eaters who pick out the clam meat will still be poisoned. Accidental poisoning from shellfish is a fairly common occurrence, especially to newcomers to coastal areas and the ignorant. The fact that the poisoning victim, if still alive for the moment, will not be able to speak assures that the accidental poisoning story is the only one heard.

For other situations, feeding the shellfish just is not practical. In cases like this, it is very useful to extract the poison from the shellfish so that it may be injected or added to the mark's diet. The process of extracting the saxitoxin from shellfish is pretty easy and results in a very potent product. No special equipment is used in the extraction, and all the chemicals used can be picked up at the local hardware store with no suspicion involved.

The extraction method given here is from an article by Helmut Muller in the *Journal of Pharmacology and Experimental Therapeutics*, volume 53, pages 67 to 89. This article is simply written, and would make good reading for anyone interested in shellfish toxin.

- The first step in extracting the poison is to dissect the shellfish. For clams, the siphons must be cut out and saved. For mussels, the dark meat is cut out and saved. Freezing is a good method to preserve the shellfish if processing to purified poison is not begun immediately after dissection. If processing is begun, the shellfish parts should be put into a blender, and an equal volume of acidified alcohol added. This acidified alcohol is made by mixing 1 ml of 28% hydrochloric acid (also called muriatic acid at the hardware store, and sold for such mundane purposes as cleaning swimming pools and cement) with 200 ml of 95% ethanol (ethyl alcohol, sold as 190 proof grain alcohol at the liquor store, or as 95% denatured alcohol at the hardware store).

It is wise to hold back a small sample of the shellfish from processing so that it may be tested for potency. This is done by feeding some of it to a particularly obnoxious dog or cat. Pound for pound, they are twice as hard to kill with shellfish toxin, so some preliminary calculations can be made as to how potent the shellfish collected are.

The shellfish-alcohol mix in the blender is blenderized for a couple of minutes, then it is mixed with vermiculite in a large bowl until a mealy consistency is achieved. The next step is to filter this mess. A large coffee filter, about a foot in diameter is perfect for the job. The coffee filter is put into a tomato juicer or some other suitable holder, and the shellfish-alcohol-vermiculite blend is put in the filter. Some more solvent (mixed up by adding 150 ml of 95% ethanol to 750 ml water and 5 ml hydrochloric acid) is worked into the shellfish mix, and the brew which filters through is the crude poison. Some more of the alcohol water-acid mix may be worked through the shellfish mush to be sure that all the poison is out of it.

The crude poison brew that has filtered out of the mussel mix is put into a glass cake pan and moved to a warm place out of the sun with good ventilation so that the alcohol can evaporate off. When the brew no longer smells like alcohol, the greenish mud that remains is purified by pouring mineral spirits into the cake pan, mixing the mud well with the spirits, and letting it sit for a couple hours. The mineral spirits will dissolve the grease out of the clam mud, and leave the purified poison sitting on the bottom of the pan. The mineral spirits are poured off, and the poison sitting on the bottom of the pan is allowed to dry. After testing to determine how potent it is, it is ready for use.

If this method sounds too hard, there is another one. It has its advantages, and it is my favorite of the two. In this method, the clam siphons or mussel dark meat is Blenderized, and then spread fairly thinly on Teflon-coated cookie pans to dry. Some mild heat is good to speed this along, but it should not be baked. When it is dry, the powder is scraped off and put in the large coffee filter. This is

then put in a tomato juicer or some similar holder and mineral spirits are poured slowly through the meat to soak out the grease and other unwanted crud. One can test to see if the process is complete by catching a few drops on a piece of glass and letting the mineral spirits evaporate. If there is grease left on the glass, the process is not finished.

When all the grease is removed, the same alcohol-acid water mix used in the first method is slowly poured through the meat. The alcohol dripping through is caught in a clean glass pan. Some effort should be made to be sure that all the alcohol is squeezed out of the clam or mussel meat before throwing away the meat.

The alcohol brew containing the poison will keep virtually forever, so long as it is reasonably acid. Some indicating pH paper comes in handy here. It is easily available from a variety of sources with no suspicion attached. The pH of the brew should be 2 to 3. Its potency may be tested in the same way as the clams or mussels were before processing. When ready for use, the brew may be evaporated down to a reasonable volume and administered to the mark. The much greater toxicity of shellfish poison when injected, and its fairly low molecular weight (about 600) suggest that this poison may be suitable for absorption through the skin by mixing it with DMSO. I suggest this as a fertile field for research by serious experimenters.

The purified poison described above is far from pure. It still contains a lot of foreign material from the shellfish in it. This unwanted material may be removed, resulting in pure shellfish toxin, but the process is a little more difficult than getting this crude material, and more difficult to obtain chemicals and equipment are involved. One can read all about it in the *Journal of the American Chemical Society*, volume 79, pages 5230 to 5235. The author is Schantz.

A quick and easy step to further purify the material is to mix about half a teaspoon full of powdered activated charcoal (available at any good pharmacy) into the alcohol solution of the toxin. After stirring it around for a few minutes, the charcoal can be filtered out

through a few coffee filters. The result is a much lighter colored mixture, once all the charcoal has been filtered out.

Care must be taken not to use too much charcoal, as it will absorb the poison also. In fact, absorption of the toxin onto acid washed Norit brand activated charcoal, and then soaking it out of the charcoal, forms the basis of the purification scheme in the article by Schantz.

Chapter Eight
TREMBLES

Several common weeds are the source of a poison that results in a mystifying and deadly disease. The little known and poorly understood disease called trembles or milk sickness is caused by eating meat or drinking milk from animals which have fed on Richweed or Rayless Goldenrod (*Eupatorium urticafolium* and *Aplopappus heterophylus*). It can also be caused by eating the weeds themselves or a purified oil from the weeds called tremetol.

These weeds are very common in wild areas east of the Rockies, and go by a variety of common names, for example: white sanicle, squaw weed, snake weed, pool wart, white snake root and deer wart. My notable lack of artistic ability prevents me from drawing what they look like here, but any good field guide will show them and the areas where they will most likely be found. Virtually every library has field guides, and they are among the most popular books in any library No suspicion is attached to checking out field guides to weeds.

The illness caused in the unfortunate soul who consumes these weeds is a puzzling thing that in many ways resembles a severe attack of diabetes. A few hours to a day after consuming the deadly repast, symptoms begin as a feeling of tiredness with pain and stiffness in the legs. The appetite is lost and vomiting follows. In no time at all, the victim is down for the count. The breath smells like acetone, and blood sugar is very low, just as in the severe attack of diabetes. The blood also becomes acidic, which is often seen with diabetes, infections and kidney failure. Death generally comes

within the first couple days of the illness, or during a relapse, which is common. An autopsy will show the liver and kidneys in bad shape, but not much else except for pancreas damage, which will fit in with the diabetes theory quite well.

What happens to the active ingredient, tremetol, once it is in the body is not known. About all that is known is that it comes out in the milk, hence poisoning from drinking milk from cows that eat the weeds. Old Abe's mom, Nancy Hanks Lincoln, is said to have died from trembles.

It is also known that tremetol is a cumulative poison, meaning that little bits taken over a long period of time are just as deadly as a lot taken at once. In fact, this is the way that most people are stricken by trembles. They drink a little bit every day of the milk from a cow that has been eating those sinister weeds with roots in hell.

It is an easy matter to extract the tremetol out of the death dealing weeds, and considering the puzzling disease that it causes, a serious student of assassination would be well advised to do so. The plant oil is much less bulky and much more appetizing than the leaves and stems and seeds of the whole plant.

To extract the tremetol out of the weeds, some fresh weeds must be collected. It will not do for them to be dried, because their deadliness disappears as the plant wilts. The plants should be chopped up into pieces less than an inch long, and put into a blender along with a couple cups of 95% denatured alcohol or 190 proof grain alcohol. After the blender reduces the mixture to an even pulp, the mush can be put into the filter basket of an Iso-II (best way to go) and some more alcohol put in the bottom of the Iso and extraction begun. Alternatively, the mush can be filtered through a large coffee filter. The plant material that remains should be mixed with some more alcohol and allowed to sit for at least a few hours with some occasional shaking or stirring. Then this brew is filtered through a coffee filter again, the alcohol that filters through being mixed with the first batch of plant juice. Finally,

some more alcohol is splashed onto the plant remains, and the last bit of juice squeezed out of it.

The greenish colored plant juice can be turned into a golden colored brew by adding about half a teaspoon of powdered activated charcoal to the alcohol solution, and stirring it in well for a few minutes. It should then be filtered through coffee filters until all the activated charcoal has been removed.

The alcohol is then allowed to evaporate off. Pouring it into a glass cake dish and setting it in a warm spot will speed up the evaporation greatly. Once most of the alcohol is gone, and the mix begins to get thicker, it should be poured into a tall glass of water with good stirring, then allowed to sit for a day or so. The tremetol will separate out and float on the water as a milky or golden colored oil. This can be collected with an eyedropper for use.

Tremetol smells like turpentine, and is about as thick as turpentine. Its taste I can't vouch for, but I would bet it is not very appealing. Tremetol is probably best served as an addition to the mark's liquor cabinet. This is only going to be successful if the mark drinks his liquor straight, because mixing the mixer in with the liquor will cause the tremetol to come out of solution and float on the drink. A scene like that would doubtlessly arouse the suspicions of the mark, and could lead to explosive situations. Very greasy foods like chili could also make a good means of serving the tremetol.

The fatal dose of tremetol is not known. This just further shows how little is known about this poison, and how far off the beaten path it is. My guess is that the fatal dose is in the 1 to 4 ounce range. Advantage can be taken of tremetol's cumulative effect by giving smaller amounts until symptoms begin to appear, then following with a larger one to put away the mark.

Chapter Nine
BOTULISM

There has been a lot of nonsense written about botulism in both the popular and underground press over the years. Entertaining stories about Pancho Villa aside, I will attempt to describe exactly how botulism germs can be grown, and their exceedingly poisonous product used for both assassination purposes and as a weapon of mass destruction.

Every once in a while, a story comes to the public's attention about some poor unfortunate(s) falling victim to botulism as a result of eating improperly preserved food. If a commercial product is at fault, a mad scramble ensues as the authorities try to get a recall in motion before more victims succumb to the tainted tasties. In these investigations, there is always a mood of tragic accident and misfortune, which is why botulism is so highly recommended as a tool for assassination. It is always assumed that cases of botulism are accidental results of eating bad food, not the handiwork of a crafty executioner.

Botulism is a disease generally caused by eating food in which the bacteria *Clostridia botulinum* has grown. The bacteria produces a most exceedingly poisonous protein, botulin, as a normal part of its growth. The poison production is the bacteria's way of claiming the particular chunk of food it is growing on as its own. Death is the penalty for any animal reckless enough to snatch it from its grasp.

Botulin is the second most powerful poison known, taking the runner up position to a poison made by an exotic strain of South

Pacific coral bacteria. The fatal dose of pure botulin is in the neighborhood of 1 microgram, so there are 1 million fatal doses in a gram of pure botulin.

The bacteria that makes botulin, *Clostridia botulonum*, is found all over the world. A randomly chosen soil sample is likely to contain quite a few spores of this bacteria. Spores are like seeds for bacteria, and can withstand very harsh treatment. This property will come in very handy in any attempt to grow botulism germs, because other germs can be wiped out by heating in hot water, leaving the spores to germinate and take over once they cool down. Much more on this later.

Another very important property of botulism germs is that they can't survive exposure to air. The oxygen in it kills them, but does not kill their spores. Whatever toxin the germs made before their demise also survives. This need to exclude air from the environment where the germs are growing is the most difficult engineering challenge to the aspiring cultivator of *Clostridia botulinum*.

Finally, all botulism germs are not created equal. There are subgroups within the species that make toxins that vary immensely in their potency. They are called types: A, B, C, D, E, F and 84. Type A is by far the most deadly, followed by type B and 84. The other ones we won't even bother to discuss. Also within a single type, there are individual differences in how much toxin a given strain will produce. Breeding and gene manipulation have a lot to do with this, and our government (and the Russkies as well) have put a lot of effort into picking out strains that make an inordinate amount of toxin. The champion as of about 30 years ago was the Hall strain, but I'm sure that they've come up with something better since then. The Hall strain of type A was able to make 300 human fatal doses of botulin per ml of broth it grew in.

Here we will explore the two major levels of use for botulin as an attack weapon: the individual or small group assassination, and the large scale assault with the poison in a manner similar to nerve gas.

Small Scale Attack

On this level of attack, the need for training in bacteriology almost disappears. All that is needed is an ability to work carefully, and a basic knowledge of sterile technique. The widespread cultivation of magic mushrooms is evidence enough that these skills can be found outside of the traditional 4 year college setting.

For this attack plan, the best approach is the one I call "the accidental clumsy canner." This line is chosen because it is so simple, and is also the way that most cases of accidental botulism poisoning occur.

The process of canning is ideally suited for getting a horde of botulism germs growing. This is because the process of canning (if not done in a pressure cooker) is one where the can of goodies is heated in a water bath with a sealant cover on the can. The contents of the can get heated enough to kill living germs, but not spores. The air in the can gets driven out, and replaced with steam. As the can cools down, the sealant cap keeps air from returning to the can and a vacuum develops. The result is an oxygen free botulism paradise.

The only reason why such home canned goodies are not rampant with botulism is that there is another factor very important to the growth and happiness of botulism germs. They are fussy about what they like to grow in, its pH, and the temperature. All of these factors are easily controlled by an attacker to give the botulism germs a happy and healthful (for them) home.

The conditions most favorable for growing a vigorous culture of botulism germs include a food source that is rich in protein, a pH that is nearly neutral (the ideal starting pH is 7.2, which is so close to neutral that indicating pH paper will show no difference between 7.2 and the neutral 7.0), and a nice warm place to grow in undisturbed for a few days. Their favorite temperature is 35° C (90° F). They can handle cooler temperatures, but warmer temps may do great damage to the culture of germs.

Now that the basics have been covered, how exactly would the "accidental clumsy canner" attack plan be carried out? The first step is to obtain some spores of the botulism germ. These are not the sort of things one picks up at the corner drug store, so a field trip is in order to obtain soil samples likely to contain the spores of *Clostridia botulinum*, preferably type A for maximum effectiveness. A lot of research has been done on the types of soil most likely to contain large amounts of spores. I can summarize the findings by saying that *Cl. botulinum* prefers dark, rich, fairly wet soil that is rich in organic matter. Sandy soil is not so well suited for *Cl. botulinum*. It is also more heavily populated in warm regions than cold ones. Finally, type A is the predominant type of botulism germ in the US, with virgin soil holding the greatest preponderance of type A germs.

The following articles will prove invaluable in fleshing out these generalities with more concrete specifics:

"Incidence and Distribution of *Cl. botulinum* in Soils of Illinois" by M.T. Jones, *Fd. Res.*, Vol. 10, page 238 (1945)

"Survey of Soils for Spores of *Cl Botulinum*" by C G. Knock, J. *Sci. Fd Agric.*, Vol. 3, page 86 (1952)

"The Distribution of Spores of *Cl. Botulinum* in California" by K.F. Meyer, *J. Infect. Diseases*, Vol. 31, page 541 (1922)

Same title for US — *ibid.*, page 559

Same title for Alaska and Canada — *ibid.*, page 595

"Spores of *Cl. botulinum* in Georgia Soil" by R.E. Morse, *Fd Res.*, Vol. 15, page 454 (1950)

"Prevalence of *Cl botulinum* in Soils of Central New York State" by E.W. Parry, *Fd Res.*, Vol. 11, page 203 (1946).

This is all very easy. It is more difficult to find soil samples that do not have botulism germs in them than to find those that do. The sample should be taken a few inches below the surface.

With several likely samples in hand, it is time to begin canning. A good home medium for growing botulism germs is greasy beef. Some cheap, fatty cuts are deboned, and run through a blender. An equal amount of tap water is added to the mush, and it is put in

narrow-mouth quart size canning jars. They should be filled nearly full, so that there is not much air space left in them. Then about half a tablespoon of soil sample is added to each jar. Two jars for each soil sample should prove sufficient. The lids are put on for canning, and the sample is shaken to mix it through each jar.

The jars are then placed in a large kettle containing boiling water, and heated for 45 minutes to one hour, just like canning tomato juice. Microbiologists call this step "heat shocking." It wipes out the living germs, leaving only the spores to survive. The purpose of the fat in the beef is to melt, rise to the top and seal off the beef from any possible air on cooling. The airless condition of the jars after the lids seal upon cooling means that only anaerobic (means they grow in the absence of air) spore-forming bacteria will be able to grow in the jars. This still leaves several species of germs besides botulism germs to contaminate the brew, but many of them, like tetanus, make poisons of their own, and so will not cause great harm.

After the heat shocking, the jars are taken out to cool. The jars are put in a warm place to grow. They should not be shaken, or in any other way disturbed, as this might cause air to get into the broth. The canning rings may be loosened as soon as they cool down, because growing botulism germs make hydrogen sulfide gas (rotten egg odor) and this gas may build up pressure and cause the jars to explode if they can't vent it off

After a few days to a week at 90° F, the jars should be just about ready The jars containing botulism cultures will be easy to recognize. The meat will have turned black, and be at least partly digested by the little devils. A foul-smelling gas will have built up in the jars, and ruined the seals on the lids of the jars.

The next step is to test the poison level in promising jars. Mice, hamsters and guinea pigs come in handy for this. Wearing rubber gloves, the lid is carefully cracked open a little bit, and a few drops of the brew are removed with an eye dropper. The brew then goes down the mouth of the unfortunate test victim. Within a few days, the animal should be dead, if it is a good batch. The delay of a few

days is due to the nature of the poison, and is not an indication of how good the batch is.

With the first test passed, the next step is to test it on a larger animal to see how potent the brew is. The signs of poisoning are sleepiness, vomiting, loose bowels, paralysis and death.

With the preliminaries taken care of, the botulin is ready for its target. A good batch should have several lethal doses of botulin per ml of broth in a jar. This small amount of foul-smelling slop can easily be disguised in a strong-tasting mixer, such as chili, or coffee. Care must be taken with the botulin that it not be subjected to boiling water or similar high temperature. Botulin is a protein, and will be denatured and made harmless by the heat.

Once the symptoms of botulism appear, the antitoxins that medical science has developed are completely useless. Whether the victim lives or dies as a result of botulism depends solely on how large a dose the victim has received, and how tough the victim is. Treatment before the start of symptoms offers some hope, so long as too massive a dose was not ingested.

In its earlier stages, botulism in humans is often misdiagnosed as such things as stomach flu, polio, heart trouble, or heavy duty booze bounding. However, by the time the later stages of the illness are reached, the doctors usually have it figured out. For this reason, the assassin does not rely on the unexplained mystery disease as his line of defense. Instead, the line taken is the one of accidental contamination of the food supply.

Large Scale Mass Destructive Attack

The large scale production of botulin toxin is an alternative to the use of nerve gases for assault on enemy population centers. This may at first surprise you, but the crude production techniques of the small scale attack and "the accidental clumsy canner" can be radically improved upon to yield mass quantities of terrifyingly potent botulin toxin.

The use of botulin has several advantages over Sarin or the V-gases as an agent for devastating enemy cities. First and foremost, botulin is several hundred times more potent than even the most potent V-gas. This huge jump in toxicity means that one reasonably large bomber plane (like a Cessna Cub) could carry a payload that would wreak havoc with an entire metropolitan area. This is because botulin is quite deadly when inhaled (even more so than when eaten) so a bomb design similar to the one for Sarin and the V-gases results in a punch rivaling that of a tactical nuke.

The second advantage of botulin manufacture over nerve gas production is that it does not use the large amounts of chemicals that are necessary for a massive scale assault with the nerve gases. This allows the attack group to evade any scrutiny that may be covering the chemicals used for nerve gas manufacture. This scrutiny is not likely to be covering the basic precursors emphasized in this work, but one never knows how pervasive Big Brother's snooping is. The recent explosion of biotechnology companies and microbreweries creates an excellent forest for the botulin producer to hide in.

A third factor to be kept in mind with botulin production is that a person can be vaccinated against poisoning by botulin. This is standard procedure for the workers in the Army's chemical research facility. Three injections of the "toxoid" provide reasonable protection against small exposures to botulin. Attempts to get vaccinated through normal medical channels could arouse suspicion, so making homemade toxoid may be called for. See *Journal of Immunology*, volume 55, pages 245 to 254, for directions. The author is C. Nigg. This compares favorably to nerve gas production, where the only protection is to avoid all exposure to the chemical.

Finally, the poison is all natural, and is made without resorting to preservatives, artificial colorings or flavors. Just the down home goodness of Mother Nature.

The basic course of action can be outlined as follows: First of all, a pure culture of only one strain (chosen for its potency) of *Cl*

botulinum type A is isolated, and then grown on a large scale following the directions that the Army chemical corps has so profusely published in the scientific journals. From these large scale cultures, the purified toxin is isolated by means of really simple chemical means, such as acid precipitation. With a large stockpile of purified botulin thusly obtained, weapon production proceeds in a manner very similar to the nerve gases.

I have a degree in biology, as well as chemistry, and so have taken a couple microbiology courses, as well as several other courses where culture technique was an important part of the class. So I believe that I am in a position to be able to give my opinion on the difficulty of this process. For me, the most difficult part would be to isolate and positively identify a colony of bacteria as *Cl. botulinum* type A. From that point on, everything would be very easy, simply a matter of maintaining anaerobic conditions in the cultures, and keeping them from getting contaminated with freeloading germs blowing by in the air. In any case, this attack plan requires the participation of someone with a background in microbiology (beyond growing funny mushrooms!).

The first step in the industrial scale production of botulin is the same as for the "accidental clumsy canner" attack plan. Some spores of *Cl. botulinum* are obtained by collecting a series of likely soil samples. Each one of these soil samples will be teeming with a wide variety of microscopic life, so the first thing which must be done is to wipe out as much of the unwanted stuff as possible.

To do this, the samples must be heat shocked to kill everything in it except the spores. Since only a few classes of bacteria make spores (including *Cl. botulinum*), this narrows down the field nicely. Sterile technique must be used from this point onward to prevent contamination of the cultures.

About one half teaspoon of dirt is taken from each soil sample, and put into a separate clean test tube. Water is added until the tube is about half full, and each one is shaken well to suspend the spores. A ball of clean cotton is put in the ends of the tubes, then they are placed in a pan of boiling water. Once they are in the water, the heat is turned off, and the temperature of the water is allowed to fall to 80° C, where it is held for about 45 minutes.

From now on, anaerobic conditions must be maintained. Some good books covering this technique are:

Diagnostic Microbiology by Bailey and Scott (Highest recommendation; it also contains formulas for all the media needed to isolate *Cl. botulinum.)*

Microbiological Methods by C.H. Collins

Isolation of Anaerobes by D.A. Shapton.

Test tube slants containing thioglycollate media are inoculated by the stab technique with the water in the tubes that have been heat shocked, and after sealing with some freshly autoclaved Vaseline, incubated for 72 hours at 36° C.

From each slant, a sample is streaked onto freshly made egg yolk agar, and incubated for two to three days at 30° C. Colonies of *Cl botulinum* have a distinctive appearance, and a unique pattern of digestion of the egg yolk agar. Some good, albeit black and white, pictures of these colonies on egg yolk agar can be found in the *Journal of Bacteriology*, volume 53, pages 139 to 146. The author is L.S. McClung.

When some promising colonies are identified, small portions of the colonies are removed with a loop or needle and smeared onto a microscope slide. A microscopic examination of the gram stained germs is made to see if they match the appearance of *Cl botulinum*. A good picture of these germs can be found in the Bailey and Scott book (fourth edition) page 186. They should also be gram stained to make sure they are gram positive.

When several good colonies of *Cl. botulinum* have been positively identified, the next step is to pick out the most potent strain for use in mass cultivation. To do this, a series of test tubes is filled with chopped meat medium (see Bailey and Scott for the formula), and each one inoculated with a different botulism culture. These tubes are incubated for 5 days at 30° C, then each one is tested to see which tube contains the most potent poison. To do this, some white mice (or similar sacrificial victims) are obtained,

and carefully measured amounts of each tube are removed. Each of these portions should be diluted about one to a hundred, and for starters, one tenth of an ml of each diluted culture injected into a separate mouse. Those cultures which yield fatal results in a few days should be tried again at greater dilution, until the most potent of them all is found. This will doubtlessly be a good producing type A strain.

With this "master race" strain chosen, it is time to gear up the production. Several larger jars are filled with chopped meat medium, and inoculated with this strain. These will be stock cultures to be used as starters for each of the large scale batches to follow. They are incubated at 30° C for 5 days, then put in the freezer.

Jugs for brewing the large scale batches must now be chosen. One gallon size glass jugs are convenient because they fit into a standard pressure cooker for sterilization, but with a larger autoclave, larger jugs greatly increase the rate of production, and reduce the amount of repetitious work which must be done.

An excellent and cheap medium for getting high yields of toxin was worked out by the guys in the Army chemical corps. They published their work in the *Journal of Bacteriology*, volume 53, pages 213 to 229. The authors are Lewis and Hill, and the title is "Practical Media and Control Measures for Producing Highly Toxic Cultures of *Cl. Botulinum* Type A." It is must reading for anyone interested in botulism. Their formula calls for use of clarified corn steep liquor because the regular stuff settles out a bunch of solids when sterilized in the pressure cooker. They made this clarified corn steep liquor as follows:

The corn steep liquor was diluted with enough water to make it thin and free flowing. Its pH was then adjusted to between 8.4 and 9.0 with 50% sodium hydroxide. Next, it was heated to boiling, and then filtered or centrifuged to remove the sludge.

With that taken care of, the formula for the medium is:

Solution A: Add 20 grams powdered milk to 180 ml of water. A little bit of 1 M NaOH, a few drops at a time with stirring, until the milk mixes in nicely.

Solution B: Mix 6 grams commercial grade glucose with a portion of the treated corn steep that contains the equivalent of 4 grams of solids (as prepared, that's about 12 grams). Dilute this to 800 ml with water.

When ready to go, mix together A and B well, and adjust the pH to about 7.5. Pour it into the culture jugs, and sterilize in a pressure cooker at 15 lbs for 15 to 20 minutes. After sterilization, the pH should be very close to 7.0. A pH meter with glass electrodes is very handy for this process. Cost when new: $200 for a cheapie. This formula makes one liter of medium, but the formula can be scaled up to fill whatever size jug is being used.

When the jugs cool down they are inoculated with about 2% by volume of an actively growing culture of *Cl. botulinum* in chopped meat medium (started from the stock in the freezer), and incubated at about 34° C. If the jugs taper at the top towards a small opening, and are filled pretty full so that there is not much of an area of liquid surface exposed to the air, pouring a bit of freshly autoclaved Vaseline on top will seal the culture. Agitation of the cultures must be avoided.

In 72 hours, the brew is ready for harvesting. The pH of the mix is now acid, which is good because the poison is not stable at alkaline pH. The Army's Hall strain regularly made 1 million mouse lethal doses per ml of this culture. It would seem reasonable to me that a good wild botulism strain should do about half as good.

The poison may now be extracted from the culture medium. Once again, the Army has published their research on the best way to do this. It is not at all hard, but can be dangerous. Protective clothing, respirators, and fume hoods are all called for.

The full details of the isolation of botulin toxin can be found in the *Journal of Bacteriology*, volume 73, pages 42 to 47. The author is James Duff. To start with, 3M HCl (about 10%) is added slowly to the cultures with stirring until the pH is between 3.5 and 4.0. This causes the poison to precipitate out and settle to the bottom of the jugs. The jug is allowed to set overnight at room temperature to complete the process, then the supernatants are removed and thrown away, and the toxic precipitant poured into a smaller jug to settle some more at 4° C (40° F). This precipitant is 25 times more potent than the original culture, and takes up 1/40 the volume. The percentage recovery from the culture is 96%.

The toxic precipitant is next washed with some water. To do this, the precipitate is diluted to 4 times its volume with water, its pH adjusted to 5.0 with diluted HCl or NaOH, the mixture stirred well, and then it is allowed to stand overnight in the fridge at 4° C.

The supernatant is thrown away as before, and the precipitate is centrifuged at 4° C for half an hour at 3500 rpm. Once again, the supernatant is thrown away, and the precipitate is resuspended in distilled water to a volume of 1/40 of the original culture volume. The % recovery in this step is 80% from the original culture, and purifies the material 2½ times from the previous step.

Now in the final purification step, the majority of the contaminants can be turned into a sludge by adding calcium chloride to the mixture. This sludge can then be filtered out. To do it, the centrifuged toxin is diluted to 4 times its volume with water and 1 M $CaCl_2$ until the $CaCl_2$ concentration is .075M$CaCl_2$, and the pH is adjusted to 6.5. This mix is filtered through filter paper at room temperature, and what filters through is the purified poison. It contains 66% of the original toxin. This high loss, and the increase in volume at this step means that a more powerful weapon can be constructed by omitting this step, and simply using the centrifuged precipitate to load into bottles for spray dispersion onto the target. Use of explosives to disperse will lead to a large loss due to denaturing of the protein. Cost of a centrifuge when new is about $600.

As an alternative, nearly pure botulin can be obtained by going on to the next step in the purification. To do this, the pH of the filtrate from the preceding step is adjusted to 3.7, the mixture cooled to -5° C, and 50% ethanol (100 proof vodka) is added until a concentration of 15% alcohol is reached in the mixture. The botulin precipitates out, and after sitting overnight, is centrifuged for 30 minutes at 3500 rpm to get the last of the product. By now, only half of the original botulin from the culture remains, the rest being lost in the purification process. For this reason, I feel that no purification beyond the original acid precipitation from the culture, washing it with water, and centrifuging it, is called for. It may not be very pure at that level, but at least the vast majority of the botulin produced will be collected. At that level of purification, it is already much more potent than the V-gases.

At a rate of production of 10 gallons of cultures per week, one person would be able to make one half gram of botulin per week. This may not seem like much, but it should be kept in mind that this is half a million human lethal doses. Greater rates of production can be obtained by processing more cultures, but that would require the assistance of a laboratory Igor.

Botulin keeps well so long as it is kept cold and out of the light. When it makes its way out into the world, more dangers await it. As was mentioned earlier, botulin is a protein and so is denatured by heat. It also can't stand basic conditions, but does not mind exposure to dilute acids. The combination of hot and basic water quickly does it in. Cold water that is neutral or slightly acid will not harm the botulin, so poisoning of water supplies is an alternative for a botulin attack. While considering this, one must keep in mind the very small percentage of a city's water supply that goes into human drinking purposes. Much larger amounts go into industrial processes, and flushing toilets. Botulin tainted water that is used to flush a toilet, is just botulin down the drain. It is also well to keep in mind the huge size of the typical reservoir. When full, they usually have enough water in them to last for a year. Poisoning the whole reservoir would require huge amounts of toxin, and would be

wasteful. Beyond that there is the water purification system for the botulin to make it past.

A more directed water supply attack may make more sense, such as contaminating a well or pipeline which serves an enemy complex. Details of this attack plan are best left to the fertile imaginations of the attack group.

For aerial dispersal, the same procedure can be used as that given for ricin in the following chapter, with the difference being that the pH of the water solution should be 6.5.

Chapter Ten
RICIN: KITCHEN IMPROVISED DEVASTATION

For those unable or unwilling to tackle the more technically demanding tasks of nerve gas manufacture or botulin culture, Mother Nature's bounty has provided a considerably more low-tech alternative: Ricin. Ricin is an exceedingly toxic protein found in the castor oil bean. One pound of pure ricin is a lethal dose for over three million people if delivered by injection. If inhaled in the form of a fine dust, it is nearly as potent. Eating ricin also produces a lethal result, although the dose must be considerably higher due to partial destruction of the poison in the digestive tract.

In mass-attack situations, ricin has one great advantage over nerve gas. This advantage is a delay in the onset of symptoms of poisoning of 12 to 24 hours. As a result, a target most likely will not realize it is under attack. Further, once symptoms do begin to appear in the unfortunate targets, it will be very difficult for investigators to back-track to the site of the original attack. These two factors combine to greatly increase the likelihood of success for the attack mission.

For assassination, ricin is similarly well-endowed with favorable qualities. There is no antidote or treatment for ricin poisoning. A lethal dose produces death, period. The long delay between administration of the poison and symptoms means that the mark will very likely not be able to make the connection to where or from whom he met his demise. Treatment and autopsy investigations are further frustrated by the fact that ricin leaves no traces of itself in

the body, and there is no test for it. A couple of hours after administration of the poison, blood from a poisoned animal can be transfused into another animal with no harm to the receiving animal. This is because all the ricin has already passed from the bloodstream into individual cells. Pretty much the only way the ricin poisoning could be identified positively as such is if mushed-up whole castor beans are fed to the mark. Then close examination of the resulting Hershey squirts will probably find fragments of the beans. A refined extract will not be detectable.

A tale from the heydays of the Cold War is instructive on this matter. Ricin was a favored assassination tool of the KGB. One person who became a target of their wrath was a defector from the Eastern Bloc who fled to England where he proceeded to make trouble for his former oppressors. To silence him, a KGB operative walked up behind him on the crowded street, and poked him in the back of his leg with the tip of his umbrella. This specially rigged umbrella then injected into the ill-fated defector a small metal ball with a hollow core packed with ricin, and a couple of holes in the ball to allow for blood circulation to dissolve the ricin and release it into the blood stream.

Within a day, the doomed man made his way to a hospital, where his condition continued to deteriorate. Given his position, he rightly suspected that he was the victim of foul play, not the flu or some bad sausage. Run whatever tests they might, however, the doctors couldn't get a clue as to what his problem was. Finally, X-rays revealed the small metal ball in his leg, and it was removed. Traces of ricin were found still on the metal, but nothing could be done to save him. Had this metal ball not been found, the cause would never have been determined.

The spectacular toxicity of ricin is due to its catalytic, rather than stuchiometric, mechanism of poisoning. The ricin protein molecule is constructed of two separate protein chains, called A and B chains. The function of the B chain is to bind to the surface of a cell, and inject into the cell the A chain. The A chain then goes from

ribosome to ribosome within the cell, inactivating each, making them incapable of producing proteins for the cell to use. The A chain isn't consumed or otherwise effected in this process, so it's possible for one molecule of ricin to kill an entire cell.

The symptoms of ricin poisoning will vary with the method of ingestion. When it has been eaten, vomiting and the runs are very prominent, along with weakness and fever. Eventually, convulsions set in, and the person dies when the area of the brain that controls breathing stops working. When the substance has been breathed in or injected, then the puking and squirts aren't as pronounced. The symptoms of poisoning by ricin are quite variable from person to person, and thus makes diagnosis of the problem very difficult.

To produce ricin, one first needs a supply of the castor beans which contain the ricin. How to best get these castor beans depends upon how much ricin is needed for the desired application. The typical concentration of ricin within the beans is in the area of 7/10 of 1%, through extraction and a series of purification steps the yield is about half of that. Smaller amounts of beans can be purchased through seed dealers, or Kurt Saxon's company, Atlan Formularies. Larger amounts should only be obtained by growing a crop. The castor bean plant will grow to maturity throughout the Southern US. It prefers fertile soil with reasonable amounts of moisture. Corn or cotton fields can easily be converted to castor bean production. A short listing of seed dealers who carry castor beans follows:

Atlan Formularies
P.O. Box 95
Alpena, AR 72611
501-437-2999

Mellinger's Inc.
2310 W. South Range Rd.
North Lima, OH 44452
216-549-9861

Joe Harris
3670 Buffalo Rd
Moeton Farm
Rochester, NY 14624

Hudson Seedsman
P O. Box 1058
Redwood City, CA 94064

Stokes Seeds, Inc.
Box 548
Buffalo, NY 14240
416-688-4300

There are a numbers of varieties of the castor bean plant (*Ricinus communis*) from which one may choose. Generally, most of the varieties have only small cosmetic differences from one another that are unimportant to the end use. However, dwarf varieties such as *Ricinus communis gibsonii* should be avoided, as the resulting small beans require too much labor to hull prior to extraction. The typical castor bean is about the size of one's thumbnail.

In addition to being able to purchase castor beans from established seed sources, it may not be too difficult or suspicious to order a reasonably large quantity through a "feed and seed" retailer. It's probably wiser not to express interest directly in the castor oil crop, which is rare in most areas, but to request castor beans as a cover crop to benefit a cash crop which is common in your area. This will only ring true if you live in a warm region of the country, however. Further, if you happen to live in or travel to Florida, South Texas or Southern California, some retailers will probably have castor beans in stock during the planting season because it is in these regions that the small castor oil industry still exists.

Hybrid varieties of castor bean are superior in respect to crop yield, disease resistance, and robust growth. They are, however, a poor choice for this project because one would want to use some or all of the beans to have a larger crop in the following year. Since the seeds (beans) derived from hybrids tend to have a low germination rate and unpredictable qualities, one is better advised to stick to truebred or inbred varieties instead. By planting the many beans obtained from planting a single bean and letting the plant mature, one can, over a period of time, parlay even a small initial acquisition of castor beans into a large crop.

In tropical regions, the castor bean plant is a robust perennial that can reach 15 feet in height, and take on the proportions of a large shrub. In areas subject to frost, it can grow as an annual. It should be planted just after the danger of frost has passed, about 2-3 inches deep in fertile well-drained soil with full sunlight. When the plant matures, it bears fruits which are capsules covered with long, soft spines. These fruits contain the castor beans.

To extract the ricin from the beans, one should first remove the hulls from the seeds. This is a very labor-intensive task, and is hard on the fingers. The hulls can be made easier to remove by first soaking the beans in a solution made up of one 12 oz. can of lye dissolved in a ½ gallon of water. The soak should last for about one hour, then wash off the beans and spread them out to dry. This will help, but it's still not easy. If any of you know of a good tool for the task, feel free to write with suggestions.

Once the hulls of the beans are removed, one is left with the soft white meat of the beans. The castor oil must next be removed from the product. Acetone is probably the best solvent to use for this purpose, as it is easily available at the hardware store and the residue quickly evaporates from the bean pulp. Hexane is also a good choice if it is available to you. To remove the oil from the pulp, first mush up the beans with a hammer, and load the bean meats into a blender until it is half filled. A plastic blender pitcher shouldn't be used with acetone as it will be dissolved. A metal

blender pitcher should be used with this solvent. Now add acetone or hexane to the mushed-up beans until the blender pitcher is just about full. Then put on the cover and blend until a smooth, milky mixture is obtained. Pour this mixture into a covered glass container, and let it sit for a few hours, then filter through a coffee filter. Squeeze out as much solvent as possible from the bean pulp, either by packing with a spoon, or even better by using a Büchner funnel and filtering flask attached to a line of vacuum. Next return the bean pulp to the blender, and add a fresh load of solvent. Blenderize the pulp as before, and filter the mixture. Once the pulp has been drained of solvent, repeat the solvent extraction. Then it should be spread out to dry in an area without much wind movement, as breathing in the dust would be bad.

When the pulp has dried of solvent, it should have a chalky, talcum-like consistency. If it still seems oily, it should be extracted again with solvent. If extraction of the ricin from the defatted bean pulp isn't going to be done right away, the powder should be stored in a freezer. Always wear a very good dust mask when handling this powder. Everything which comes in contact with the bean powder should be washed very thoroughly or destroyed.

To extract the ricin from the pulp, I direct the reader to several references. All of them are relevant. See US patent 3,060,165. Also see *European Journal of Biochemistry, Volume 105*, pages 453-5 (1980) and *Biochemistry, Volume 12*, No. 16, pages 3121-26 (1973). An article from *Toxicology, Volume 2*, pages 77-102 (1974) is also interesting. For many examples of how not to go about this procedure, see *The Weaponeer* and *The Survivor* by Kurt Saxon. His bumbling and fumbling of the extraction and isolation is almost comical, and very instructive. He ultimately gave up, since he was only able to produce small amounts of a weak product. I really shouldn't make fun of him, since his formal chemistry training is obviously rudimentary. Rather, the model which will be followed here is that of Dr. Albert Hoffmann, the discoverer of LSD, and the experimental tactics he used to isolate

and find the molecular structure of psilocybin, the active ingredient of funny mushrooms. Dr. Hoffmann was able to far outpace his competitors in the race to isolate and identify psilocybin because he and the members of his research team were willing to taste-test their extracts to assure that the active ingredient was being isolated. This idea never occurred to his competition, as they were too chicken shit and too tied-up with the establishment to contemplate such a course. Obviously, taste-testing one's extracts will bring this project to a rapid and fatal conclusion. For this job, one should recruit a series of white lab rats, available at the pet store as food for your boa constrictor. This project will keep him quite hungry.

The extraction of ricin from castor bean pulp is a pretty standard protein extraction, very similar to procedures used to extract and isolate other proteins from a plant matrix. The first and most important point to keep in mind is that the extraction must be done at refrigerator temperatures. This prevents other enzymes extracted out of the bean from degrading the product ricin in solution. Exact control of the pH during extraction doesn't appear to be so crucial to success, as the *European Journal of Biochemistry* article used full-strength vinegar (5% acetic acid) as the extractant. This gives an extraction pH of under 3. Both the US patent and the *Biochemistry* article preferred a pH of 3.8 to 4, with the *Biochemistry* article using dilute vinegar to reach that pH, and the patent using dilute sulfuric acid. I am mildly suspicious of the patent, as I feel it may contain some deliberate misinformation. Beyond that, it was filed in 1952, and kept secret until 1962, so their procedure represents antique technology. The procedure followed here is a melding of the three procedures.

The defatted, solvent free bean pulp obtained from one kilo of castor bean meats (it now weighs considerably less than a kilo with the oil removed) is suspended in 4000 ml of distilled water cooled to about 4C (refrigerator temperature). Then, with stirring, add white distilled vinegar until the pH reaches 3.8 to 4. A good, accurate, properly calibrated pH meter is best for measuring pH.

The beans must be thoroughly defatted to get good extraction, so don't skimp on the solvent in the previous step. The oil would act to prevent good wetting of the bean pulp and prevent extraction. Now let this mixture sit in the fridge overnight. When morning comes, pour it into a blender, and while it's still cold, grind it hard, at maximum speed, for a minute or two, then return it to the fridge, where it should be magnetically stirred for 24 hours. Place a layer of cardboard between the stirrer and the product to prevent heat transfer from the stirrer.

Now it is time to separate the vinegar extract from the bean sludge. Filtration won't work very well, because it's just too gooey. Centrifugation is far superior. The articles recommend a centrifuge speed of 10,000g for 30 minutes. The extract must be kept cold. Work outside in the winter, or in a walk-in freezer in order to maintain proper temperature. The clear-colored vinegar extract should be separated from the centrifuge-compressed bean sludge (termed a pellet), and stored in the refrigerator. The pellet should then be extracted with one more portion of dilute vinegar at pH 3.8 to 4 at a temperature of 0-4° C with stirring overnight. This extract should then be similarly obtained by centrifugation. The US patent claims that the extract can be separated from the bean sludge by filtration, if a filter aid such as Celite 503 is added to the bean sludge-vinegar mixture to the extent that it is about 10% by weight of the defatted bean meal used. Your comments on this variation are welcome.

Now the combined dilute vinegar extracts, while still kept cold, are gradually brought to 100% saturation with solid ammonium sulfate. This requires about 70 grams of ammonium sulfate per 100 ml of extract. It should be added slowly, with stirring. This precipitates the ricin out of solution. Once saturation has been reached, keep the solution overnight in the refrigerator.

The precipitated crude ricin is now collected by centrifugation. The article recommends 23,000g for two hours. The product is the pellet. Remove the supernatant vinegar, collect the pellet, and dry it

under a vacuum. Now our animal friends come into play to test the extraction process. Feed some of them bean sludge residue to test for completeness of extraction. Feed some of them supernatant vinegar to test for completeness of precipitation, and feed some of them your crude product. Varying degrees of dilution are called for to judge just how much of this very potent poison has been wasted, and how much has been captured.

This vacuum-dried, crude ricin is likely to be good enough for most practical purposes, however, all of the articles move onward to get pure ricin. The scientific articles use chromatography, while the patent uses another extraction and precipitation step. Let's follow the patent here. Suspend the still-wet crude ricin pellet in three times its weight of distilled water, or suspend the vacuum-dried ricin in 30 times its weight of distilled water. The water should be cold, and kept cold. Now dropwise, with strong stirring, add 5% by weight sulfuric acid solution until a pH of 3.8 is reached. Stir for about an hour, then filter. Then to the filtrate, dropwise add 12% sodium carbonate solution until the pH rises to about 7. Now the pure ricin can be precipitated by slowly adding a solution made up from two pounds of sodium sulfate dissolved in 10 pounds of water to the neutral ricin solution until the amount of sodium sulfate added equals 20% of the original weight of the neutral ricin solution. For instance, let's say you have about a pint of this ricin solution. It weighs about a pound. You will want to add sodium sulfate solution to it until you have added about 90 grams of sodium sulfate.

This requires adding about 540 ml of sodium sulfate solution. Stir in the cold for an hour or so to complete precipitation, then filter. The filter cake should be rinsed with some more of the sodium sulfate solution to remove more non-toxic proteins. Just one good rinse is called for here, as more does no good. Finally, vacuum dry the filter cake.

This produces a product which is about half pure ricin, and half solid sodium sulfate. The sodium sulfate does no harm to the ricin.

However, for use as an airborne weapon of devastation, it's necessary that the size of the ricin particles be reduced to a very small size, on the order of a few microns, which is similar to smoke particles. The small size is needed so that the particles remain airborne, rather than settling out as dust. Also, the small size is needed to make sure that the particles get all the way down into the lungs, rather than being caught in the snot filled slime that covers one's air passages.

The patent addresses this problem, and says that the best results were obtained with air grinding. Putting the dried ricin into a blender and letting the blades whip it up and grind it would seem to me to be to be analogous. To prevent detoxification of the ricin, it must be quite free of water when it is ground, and kept cold. They also suggest ball-and-hammer milling of dried ricin in a slurry of CCl_4. Keeping it cold is also important here. Spray-drying a 20% solids slurry with CCl_4 was also used. See the patent. Once ground, all one needs is a fan to propel airborne death on its way to the target.

A really good method for getting a small ricin particle size can be used if the plan is to spray the enemy territory from some height, as from the top of a reasonably tall building or from a low-flying plane. This method is to dissolve the ricin into water adjusted to pH 4 with acetic acid or vinegar. No more than one gram of ricin should be dissolved in each liter of water. Then when this mixture is sprayed from a reasonable height, the water in each droplet will evaporate as it falls through the air, leaving a tiny particle of ricin to float on the wind. Naturally, the use of this plan requires that weather conditions be favorable for evaporation of the water droplet.

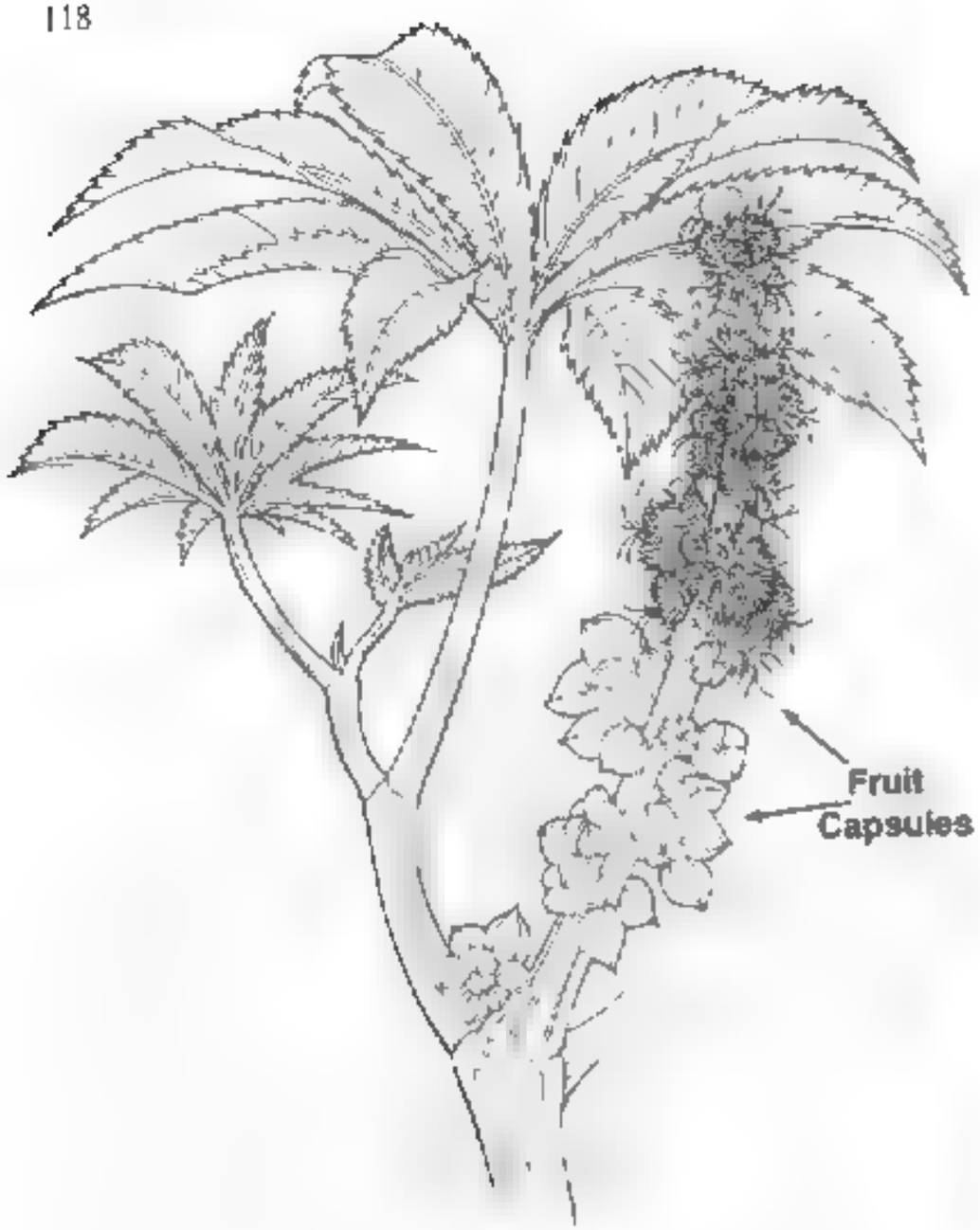
This simple procedure works well. One caveat, however: this solution will begin to grow a bustling bacteria population in no time flat. Within a day or so, floating strands of slimey gunk will be in solution, ready to plug up the sprayer. Worse still, the bacteria may

well be able to digest the ricin, rendering it harmless. For this reason, the solution must be used quickly.

Happy hunting!

***WARNING:** Possession of refined ricin will likely get you a life term! Beyond experimental runs to perfect your technique, these procedures should only be done under war situations. If stockpiling must be done, it should only be in a very secure place, with freezing temperatures to assure freshness of the product.*

Castor
Seed or Bean



Castor Bean Plant

Chapter Eleven
DELIVERY TO THE TARGET

The successful delivery of a poison to its target is an often overlooked, but vitally important aspect of successful poisoning. Many well planned attacks have fallen apart because the method of delivery and its consequences have not been thought through. The means of delivery chosen is of such importance that I believe that it is just as vital to the success of the mission as the choice of the right poison. Here we shall explore the methods used by the masters, and the mistakes made by bumblers.

As in many areas of endeavor, the first and foremost rule is: "know the target." A knowledge of the habits and likes and dislikes of the mark is indispensable for successful delivery to the target. For example, consider the case where an attacker does a "black bag" job on the mark, breaking into this house and spiking a bottle of whiskey with poison. If it turns out that the mark does not drink whiskey, and was keeping it around for guests, the attack will have been a failure, unless the object was to frame the mark for the poison deaths of his guests.

As could be inferred from the above paragraph, in many cases a "black bag" job is often called for to deliver a poison to the subject. The object, naturally, is to plant the deadly substance in some edible that the target is likely to consume within a reasonable period of time. Here the ethical attacker will take care to try to make sure that only the intended target will be exposed to danger. The death of

innocents is not only useless, but will turn public opinion against the attackers.

An alternative to the black bag approach is to have the item mailed or delivered to the mark. This route has the advantage that it eliminates the dangers of burglary inherent in the black bag technique. It has the drawback that it may not be very believable to the mark, and may arouse his suspicions. The typical mark is not likely to be receiving gifts from anyone. Some ingenuity on the part of the attacker can overcome this. For example, on birthdays or holidays, a package may arrive from out of town family or business associates. Alternatively, a promotional giveaway can be concocted for some new product.

Regardless of the delivery method chosen, the choice of edible to contaminate is crucial. For the mark who has a taste for "controlled substances," poisoned dope is made to order. The coroner will detect the drugs in the body of the mark, and the newspapers will have a field day with another "killer dope" story. Ricin is a really great adulterant for coke. Its production is well described in this book. For poisons with a strong taste, booze may be the best mixer for them.

A point of great importance is for the poison to be completely consumed by the mark. It would not do for half a dozen poisoned cookies to be sent to the mark, and only have half of them eaten. It is much easier to detect a poison in such an item than in a body. Attackers wishing to keep the cause of death a mystery will load the full dose into one serving. This works best if the poison has a time delay effect, such as, for example, the jequirity bean.

Important point number two for the successful poisoner is to guard against fingerprints. Very good fingerprints can be lifted off paper, so all packaging is done with great care. Typewriting can also be traced back to a particular typewriter, so hand printing is generally employed.

Point number three for the successful poisoner in delivering the item is to have no contact with any delivery service. They may remember faces. The U.S. Mail is much preferred in this respect,

because all one needs to do is apply a bunch of stamps and drop the package into a mail box. Anonymity is a scarce commodity these days.

The final, and most important point for successful poisoning is silence. More attackers mess up here than at any other point. For some reason, they feel an overpowering urge to boast to an associate or to confide in a spouse. Neither can be trusted. Today's spouse can be tomorrow's divorce enemy, and star witness. For this reason, the old Sicilian saying, "Silence is a friend which will never betray you," is time tested wisdom.

Chapter Twelve
TIME DELAY POISONS

The time delay poisons are the really sneaky members of the unholy family I have put together here. They are like time bombs with very long fuses that the mark carries around inside of him, until finally his time runs out and he is destroyed in a burst of its deadly fury. These poisons are for the patient attacker. Generally, the symptoms leading to death do not show up for at least a few months, and often not for over a year. The death suffered by the target is usually miserable and prolonged. Cancer is their usual result.

The time delay poisons are also the safest ones for an attacker to use. By the time the mark begins showing symptoms of disease, all traces of the poisons will have long left him. A general tox screen is not part of the standard treatment for cancer anyway. (Maybe it should be!)

One member of the time delay poison family, industrial grade rapeseed oil, has already been covered. It is so fiendishly effective that it deserved a section all to itself. The jequirity bean is almost a time delay poison, but its one to three day latent period is a little too short for it to be included in this section.

One poison that almost everybody would guess would be in this section is not. That poison is dioxin. There are a few reasons for this. First of all, the stuff is not commercially available, and so is next to impossible to find. Making the stiff is not that easy, and is filled with dangers that make this route unreasonable. Finally,

recent cancer research suggests that dioxin is not as effective in causing cancer as was previously believed.

This problem of commercial availability is a common one with very effective cancer-causing chemicals. I'm not sure whether the manufacturers are concerned about the health of the public, or if they lack the courage to make the stuff themselves.

An easily available and fairly effective carcinogen is o-toluidine. This stuff had its main uses in the dye industry, where they noticed that people exposed to it developed cancers at an alarming rate. The most effective way to cause cancers with a chemical like o-toluidine is to feed smaller amounts over a long period of time rather than a lot all at once. As a consequence of this, the most effective way to deliver such a substance to the mark may be to dissolve a few grams of it into the liquor cabinet of a moderate drinker. A real booze hound may go through the whole supply in one evening of bingeing, so some other route would have to be considered for the heavy drinking mark.

Several other equally effective carcinogens are available on the market. Any one of them, or a combination, would make a fine addition to the mark's diet. They are: propiolactone, 4-nitrodiphenyl, N-nitrodimethylamine, beta-napthylamine, dimethyl-aminoazobenzene, dichlorobenzidine, benzidine, 2-acetylamino-fluorene and 4-aminodiphenyl. A few grams of any of these substances given over a period of a few months will likely result in cancer (especially cancer of the bladder) in the near future. Given the lack of progress in "the war on cancer," a slow and ugly death is the most probable ending.

Nature provides an alternative source of carcinogens with the important advantage of being freely available to anyone. No paper trails of chemical purchases to trip up would be assassins are involved in the use of natural carcinogens.

A coarse hardy fern called bracken (Pteridium aquilinum) or brake grows in swampy and wild areas of North America and the British Isles. It is especially common in Canada, where the moose

and other wild animals have learned to avoid eating it. This instinctual wisdom springs from the fact that the bracken fern contains a potent carcinogen. Eating bracken over a period of time will doubtless lead to a case of cancer. For most targets, it is impractical for an attacker to add fern leaves to the mark's diet. In these cases, it is necessary for the attacker to extract the active ingredient from the fern leaves, and use the extract to contaminate foodstuffs or liquor supplies of the mark.

To extract the carcinogen from the bracken, one follows the exact same procedure used to extract tremetol from the white snakeroot weed. It should be borne in mind here that the extract is not a pure substance, but rather a mixture of substances. One of the contaminants has the nasty property of causing blisters, so small amounts of bracken extract must be used per feeding to avoid upsetting the mark's digestion too greatly, and thereby causing alarm and suspicion. It is possible to remove the contaminant, but the process involves such things as chromatography columns, which are beyond the scope of this book.

A little used but highly effective time delay poisoning technique is to lace the victim's food with radioisotopes. The only example of this technique being used that I am aware of involved a defector from the Eastern bloc who was done in by the KGB. The KGB went hog wild on the dosage to their victim, and he died from radiation poisoning. Even this crude method left investigators puzzled for a long time, and it was only because of the intense effort given it due to who the victim was that radiation poisoning was diagnosed. Doctors don't generally give their patients the once over with Geiger counters.

When smaller amounts of radioisotopes are used, severe radiation poisoning does not result. Instead, if it is an isotope of an element that belongs in the body such as phosphorus (best isotope: P32) or calcium (best isotope: Ca45) it is picked up by the body (after all, they are chemically identical to the regular isotope) and in the case of calcium or phosphorus, used to make bone.

Once there, their radioactivity causes the surrounding tissues to become cancerous. The most likely result is leukemia, since white blood cells are made in the bone marrow.

Radioisotopes such as P32 are not so difficult to obtain as one would at first suspect. Hospitals go through large amounts of these supremely useful substances to treat cancers and as diagnostic aids. A resourceful attacker would have little trouble removing these little gems from the premises. The key to a successful action along these lines is to know the procedure and act like you know what you are doing.

For those attackers who lack the gall to walk into an American hospital and walk out with radioisotopes, there is the option of foreign procurement. Other countries, especially underdeveloped ones, are not so fussy about how their hospitals secure and dispose of their radioactive wastes. The recent case comes to mind of the Brazilian hospital that gave their used radioisotopes to the local junkman. The ever curious and playful natives promptly proceeded to rub the "magical" glow in the dark stuff all over their bodies, resulting in several deaths due to radiation poisoning. I have no doubt that a determined and resourceful attacker could go to such a foreign country, land a few disposal jobs with the local hospitals, and end up with a nice little stash of nuke waste to bring back home with him.

Finally, for those "do it all at home" types, there is another method for getting one's hands on radioisotopes. It is a technique called neutron activation. This is a process whereby ordinary nonradioactive materials are bombarded by neutrons, which are captured by the atoms which are thusly converted into radioactive isotopes. All that is needed to proceed with this method is a source of neutrons.

Howard Morland, in his book *The Secret That Exploded*, describes an atom bomb trigger made by GE that shoots a stream of high energy neutrons at the bomb core to start fission. He also tells of a similar device available on the open market, used by oil

drillers. Chemists require a similar device as well, for use in preparing samples for neutron activation analysis. (At least those who don't have a reactor nearby do!)

Whichever source of neutrons is available, radioactive isotopes of Calcium and Phosphorus can be made by packing a few hot water bottles full of calcium phosphate, and placing them in the path of the neutron beam. The material in the bottles should be wetted with some water to help slow down the neutrons and make their capture more likely. The area surrounding the machine should be shielded with a series of large aquariums filled with water. The use of water here is important because neutrons are best stopped by water, wood, cement, and other substances made up mostly of the lighter elements. Metals like lead are nowhere near as effective. It is also of great importance that the operator keep as much distance between himself and the operating machine as possible to prevent radiation poisoning from happening to the poisoner.

After a few days to a week, the machine should be turned off, and a sample carefully removed from one of the hot water bottles. It should be as much as the attacker can deliver to the mark within the next week or so. It should be carefully placed in a glass bottle for safe keeping until delivered. The use of rubber gloves and heavy clothing will protect the person handling the material during the transfer. A thorough shower afterwards is a must.

After the transfer is complete, the machine may be turned back on to continue more radioisotopes. The half life of P32 is 14.3 days, and Ca45 is 80,000 years, so the material must be used quickly to get the full advantage of the P32.

An alternative method of using the neutron source would be to simply bombard the mark with its neutrons. This suffers from several logistical problems. For starters, the attacker must get close enough to the mark, for long enough periods of time to give the target a lethal dose of neutrons. The attacker must also be in a position where the neutron beam will not be blocked by absorbent materials like wood, cement, plaster, etc. Finally, the attacker must

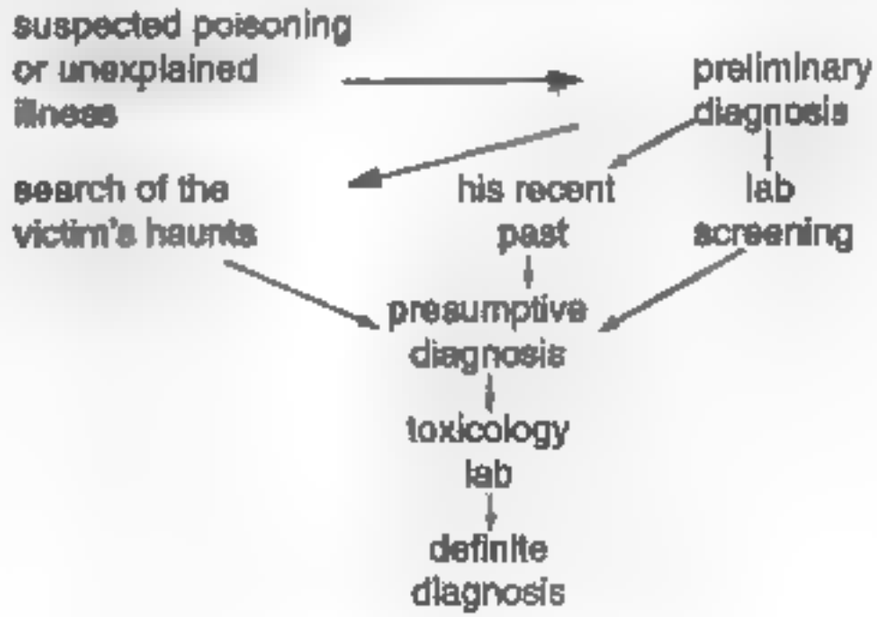
deal with the problem of arousing suspicion by carrying around a neutron generator after the mark.

For those interested in commercially available carcinogens, (or easily made ones), see *Industrial Carcinogens* by R.E. Eckardt (Grune & Stratton, 1959).

Chapter Thirteen
AUTOPSY!

In most states, the law calls for an autopsy when death occurs unattended by a physician, or occurs under violent, unusual, or sudden circumstances. The goal of the autopsy is to find the cause of death of the dear departed. In many cases, such as when the corpse has his head blown off, this is a simple matter. However, when there are no marks on the body, poison is a possibility the coroner (or medical examiner, depending on the state) must consider.

When confronted by a body or person suffering from suspected poisoning, the following series of events is par for the course for those investigating.



So first a quick diagnosis of the victim is made, based upon the symptoms he is showing, or the condition of the body. This is followed by investigation of what he has been doing the past few days, and where he has been. At the same time, a quick screen is done in the lab, with the idea of eliminating whole classes of poisons as possible culprits. With this information, an educated guess called the presumptive diagnosis is made. This presumptive diagnosis can be a whole series of possible culprits that come to the mind of the coroner. The next step is to send samples of the departed one to the toxicology lab, where each of the possibilities is checked, one at a time. This potentially very time consuming process should ultimately lead to a definite diagnosis when one of the tests at the toxicology lab turns up positive.

In theory, under ideal circumstances the above outlined procedure is supposed to lead to a sure diagnosis, finding the cause of death every time. In the real world, things don't work so smoothly. The human body, or any living thing for that matter, is not so simple. It is not like a watch or some other machine where you can point to the broken part and say, "Ah, hah!" Living things often continue to live long after the condition of their body should dictate that they be dead. The opposite is true as well; people sometimes just die for no good reason, or at least before their time.

To cope with this, coroners try to fit the body they are dealing with into one of five groups:

In the first group, the autopsy shows organs damaged to the point that life is impossible, and the death is explained beyond any doubt. A couple of good examples of this type of damage are brain hemorrhages and plugged heart arteries: The only big question to be answered in these types of deaths is what brought on "the big one" at that time. A coroner will hardly bother doing chemical analyses for poisons in a case like this. Believe it or not, these easy cases account for only 5% of all natural deaths.

In the second group, the autopsy shows damaged organs, but they aren't damaged enough to definitely account for the death.

Examples of this type of damage include chronic lung or heart disease. In these cases, the coroner will not generally bother with doing a chemical search for poisons unless he finds remains of pills in the stomach, smells suspicious odors in the body, or notes a seared stomach lining. About 85% of all deaths fall in this category.

The third group has some damage to his organs, but the amount of damage is not such that death should have resulted from it. These are guys who get their call before their time, like the guy who falls over dead getting into shape. In these cases, the coroner should dig into the guy's past, and do a search for poisons before calling the death natural.

In the fourth class of deaths, the victim dies of a disease that leaves no physical traces to be found at autopsy, such as epilepsy. A chemical search is done in these cases to rule out drug overdoses before calling the death natural.

Finally, in the fifth class of deaths, the cause just can't be found in spite of all the best efforts of the coroner and his staff. These mysterious deaths account for a few percent of all deaths.

It goes without saying that the goal of the successful poisoner is for the death to fall into one the natural death categories, most likely classes 2, 3 or 5. To do this the poison used must not leave obvious signs on the body, and must be sufficiently exotic that the people doing the investigation do not think of looking for the culpable substance involved. To quote from *Guidelines for Analytical Toxicology Programs*, page 54:

One must recognize that a toxicological survey cannot be universal. Following exclusion of commonly abused drugs and substances available in the decedent's home, place of employment, and social milieu, one is usually forced to say that the studies are negative. Unless further investigation suggests the responsibility of some chemical agent which had not been previously considered, additional toxicological procedures usually prove to be blind thrusts with scant chance of providing illuminating results.

A further point must be emphasized here. The introduction and widespread use of an analysis machine called the gas chromatograph-mass spectrometer has revolutionized this process of looking for poisonous substances in cases of suspected poisoning. Formerly it was necessary to look for poisons one at a time. This was a very time consuming process. With the GC-mass spec, three samples (an acidic extract, a basic extract, and a neutral extract) run through the machine will detect the vast majority of poisons. Alkaloids such as aconitine are easy prey to this machine. For this reason, they are all "bad poisons." A simple rule of thumb with the GC-mass spec is that if the substance can be vaporized without breaking it down, it will be detected.

The search for the inorganic poisons has also been made considerably easier since the introduction of another machine called the atomic absorption spectrometer. It never was that difficult to find these substances, but the A-A has made the process much faster and easier.

As you can appreciate from the above, successful poisoning is a much more difficult task than in the days of Lucrezia Borgia. The poisons chosen must make it past the GC-mass spec and A-A without detection. Once past them, the poisoner will very likely have smooth sailing because the great power of these two machines has the natural effect on the analyst of lulling him into a state of complacency. If they don't show anything, there must be nothing there, right?

One class of poisons which do not show up on these two machines are the proteins. These are some of the best poisons. Examples of this class include botulin, ricin, abrin found in the jequirity bean, and the poison found in the Destroying Angel mushroom. Alone in this group, the mushroom poison is a bad one. Upon autopsy, the man (ghoul) doing the cutting will see a yellow, fatty liver in the victim of the mushroom. The gruesome appearance of the liver should shout out to the ghoul that one of only a few poisons is involved i.e., yellow phosphorus, carbon tetrachloride or

Destroying Angel mushroom. The most subtle of this group of poisons are ricin and abrin, because they work by inhibiting protein synthesis in the body. They do this by inactivating the 60S sub-unit of the ribosomes, and preventing elongation. Elongation is the key step in protein synthesis, where additional amino acids are hooked onto the growing protein chain. Without elongation, all protein making comes to a stop, and death follows in a few days when the supplies on hand in the body run out. This is much too subtle to be detected in the typical autopsy.

The second major class of compounds which are good at evading detection are quaternary ammonium compounds. The prime example of this class of substances is the CIA shellfish poison, saxitoxin. These not only elude the GC-mass spec, but I know of no good test whatsoever for detecting these substances. Even if one were to be invented, it would not likely be very useful, since the body is naturally loaded with this class of substances anyway. Quaternary ammonium compounds are made by reacting an alkyl halide, such as methyl iodide, with a tertiary amine like strychnine. I know of no work which has been done in the area of alkylating tertiary amine alkaloids to quaternary ammonium compounds to see if they retain their poisonous properties. This would seem to me to be a fascinating field of study for the serious experimenter. The reaction is done by dissolving some of the alkaloid free base in ether, and then adding slowly an equal volume of alkyl halide such as methyl iodide to it. After letting it sit for a few minutes, the ether and excess alkyl halide are removed under a vacuum, and what is left is the quaternary ammonium compound.

A third major class of difficult to detect poisons are those substances which are so poorly studied that it is just not known what they are, or what happens to the poison in the body. A fine example of this class of poisons is the White Snakeroot. Of course, pioneering work could be done on the White Snakeroot tomorrow to answer all these questions, but it would still take a few years for the

information to filter down to the average working stiff in the toxicology lab.

Finally, the fourth class of difficult to detect poisons are those which are either destroyed in the body after they do their work, such as phosphine, or those which have such a long time delay, like the carcinogens, that they are long gone from the body by the time the mark gets sick. The usefulness of this class of poisons is limited only by the patience and resourcefulness of the attackers.

There are several good books on autopsy procedures for detecting poisons. If you are really into how autopsies are done, these books should answer most of your questions. (Autopsy is Pugsly's favorite game.) I must forewarn you that a good knowledge of chemistry is a must for understanding these books. They also deal almost exclusively with drugs and really crude poisons. That is because almost all cases of poisoning are accidental or suicides. Those few cases of poisoning that are homicidal are generally so crudely done that they are obvious from the start. Even so, these books will reveal a lot about the limits of modern poison detection technology. My recommended reading list is:

Poison Detection in Human Organs by Alan Curry (A real classic, but a bit dated.)

Guidelines for Analytical Toxicology Programs by Jerry Thoma

Handbook of Analytical Toxicology by Irving Sunshine

Manual of Analytical Toxicology by Irving Sunshine

Clinical Toxicology by Clinton Thienes

Chapter 14
CHEMICALS

Virtually all of the chemicals mentioned in this book have a wide variety of uses in science and industry, and are very easy to obtain either by directly purchasing them or through theft from the industries that use them. None of the chemicals mentioned in this book have any major use in manufacturing drugs that I know of, so it would probably be safe to order them from mail order outfits.

An exception to this general rule of easy availability would be the chemical carcinogens. Joining this group of harder to openly purchase chemicals are the cyanides, and arsenic trioxide. They have such widespread industrial uses, however, that theft from industry is an easy option.

What follows is a listing of the chemicals mentioned in this book, in the order they appear in the book, along with their mayor uses.

NaF — sodium fluoride, used to fluoridate water and preserve blood samples.

$NaSiF_6$ — sodium fluorosilicate, an antiseptic, insecticide, laundry soap ingredient and enameling agent.

NaCN — sodium cyanide, used in electroplating, case hardening metal, fumigation and to extract metals. For example, it finds use in recovering silver from photographic films, especially X-ray films.

$HgCl_2$ — mercuric chloride, used to preserve wood, and museum specimens, tanning, rat poisons, embalming, purifying gold and photography.

As_2O_3 — arsenic trioxide, used in manufacturing glass, leather and pigments. It is also an ingredient in lead shot.

KCN — potassium cyanide — used in gold extraction, photography and cleaning solutions.

Phosphorus — used in military incendiaries and fireworks.

Formic Acid — a reagent for nitrates in water. Also used to analyze essential oils and in tanning and leather making.

Sulfuric Acid — almost every manufacturing industry uses this chemical. A very common example is the electroplating industry.

Phosgene — its most common industrial uses are to make polyurethane resins, and in dye manufacture.

Carbon Tetrachloride — dry cleaning fluid and as a fire extinguisher.

H_20_2 — hydrogen peroxide, used as a bleach, an oxidant for fuels where air is not available (like subs), a disinfectant, and antiseptic.

HF — hydrogen fluoride, used to etch glass. I believe the result is frosted glass.

Benzidine — reagent for sulfates and blood.

O-toluidine — making dyes.

B-napthylamine — making dyes.

Calcium phosphate — a dietary supplement for calcium and phosphorus.